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PHYSIOLOGY AND PATHOLOGY

OF THE

SYMPATHETIC SYSTEM OF NERVES.

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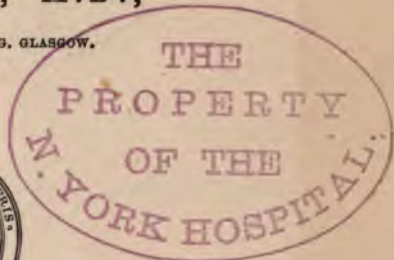
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PHYSIOLOGY and PATHOLOGY of the SYMPATHETIC SYSTEM OF NERVES.

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Part I.—PHYSIOLOGY.

Cervical Sympathetic Nerve. I. Experimental Observations on Animals with Results. A. Oculo-pupillary Branches.—The experimental investigation of the functions of the Cervical Sympathetic Nerve begins, as is well known, with Pourfour du Petit,† who, in the year 1727, showed that it exercises considerable control over the movements of the pupil.

These facts were again discovered by Biffi,‡ in 1846; he found that division of the cervical sympathetic in rabbits and dogs resulted in *contraction of the pupil*, while irritation of the peripheral end of the divided nerve produced *dilatation*. We know, further, that the pupil, contracted by division of the sympathetic, is capable of being again slightly dilated by means of mydriatics, such as atropine (Budge,§ Valentin||). The explanation of this observation, according to the general acceptance, is that in the course of the cervical sympathetic certain branches are given off which supply the musculus

* It should be stated that it was for this Essay that the Astley Cooper Prize for 1877 was originally awarded to Drs. Eulenburg and Guttmann—a decision, however, which was subsequently overthrown on the technical ground that the paper was the work of *two* authors, and not of one only—as the terms of Sir A. Cooper's will seem to require. This essay having been handed in in October, 1876, there are no references to any papers on the subject written since then.

† "Mém de l' Acad. des Sciences," 1727, p. 1.

‡ Intorno all' influenza che hanno sull' occhio i due nervi grande simpatico e vago.—"Diss. Inaug." Paris, 1846.

§ Bewegungen des Iris.—"Braunschweig," 1855.

|| Versuch einer physiologischen Pathologie der Nerven, ii., p. 154.

dilatator pupillæ, and that irritation of these produces mydriasis, and paralysing them by division *paralytic myosis*.

As regards the origin of these fibres, Budge* first pointed out that they issue from the spinal cord, for the most part from the region extending from the point of exit of the sixth cervical to that of the second dorsal nerve, which region he accordingly named the *centrum cilio-spinale inferius*.

In the same way also Claude Bernard placed what he called the "*Centre oculo-pupillaire*," at the level of the first and second dorsal vertebræ.

On the other hand, Salkowski† has lately asserted that the *Centrum Cilio-spinale* must be situated *above the atlas*, as its excitability is not abolished in blood-poisoning by suffocation—(interruption of artificial respiration in rabbits poisoned with curare)—when the cervical portion of the spinal cord is cut through, even when the incision is made above the level of the atlas.

Thus it is certain only that the zone of the spinal marrow, the wounding of which has an influence on the sympathetic pupillary branches, does not extend downwards below the level of the second dorsal vertebra; but at the present time its upper boundary cannot be specified with certainty. Besides the already-mentioned principal centrum, Budge‡ accepted a second, (*centrum cilio-spinale superius*), situated higher in the spinal marrow, which was stated to be connected with the hypoglossus by a communicating filament.

According to experiments just published by Brown-Séquard, to which we shall again refer—the accuracy of which, however, we did not find confirmed to the full extent—we may assume that the sympathetic pupillary branches probably terminate on the surface of the brain.

The connection between the *centrum cilio-spinale inferius* and the sympathetic takes place, as Budge and Claude Bernard have shown, through the communicating branches of the anterior roots of the spinal nerves. Division of these acts on the pupil in the same way as section of the cervical sympathetic itself.

Voisin§ has stated that, besides the connection by means of the communicating branches, still another exists;

* L. c. p. 108, ff. *Comptes rendus*, T. xxxvi

† "Dissert. Königsberg," 1867. "Centralblatt für die Med. Wissenschaften," 1867, No. 31.

‡ L. c., p. 128.

§ "Gaz. des Hôp.," 1863, No. 10.

certain filaments of the emerging spinal nerves surround the vertebral artery, anastomose with the carotid plexus in the cranium, and so reach the sympathetic root of the ciliary ganglion. That statement, however, is disproved by the following experiments performed by us:—The cervical sympathetic of a rabbit was divided above the spot of entrance of the nerves of the iris rising from the centrum cilio-spinale inferius; then, the edge of the transverse process, bounding externally the transverse foramen, having been nipped off with small sharp forceps, the vertebral artery was exposed and isolated as high as possible towards the foramen magnum, and subjected to the powerful stimulation of an induced current. The pupils did not become in the least dilated; the attempt was equally unsuccessful when the sympathetic was not previously divided, and also in newly-killed animals, in which the oculo-motorius no longer responded to stimuli, whilst irritation of the sympathetic still produced dilatation. Further, no contraction occurred when we cut through the vertebral arteries, or divided with bone forceps the lateral walls of the bodies of the vertebra, together with the contents of the transverse foramen down to the medullary canal. Voisin's assertion can therefore raise no claim to our consideration, especially also as it is quite unsupported by descriptive anatomy.

Some authors (Grünhagen, Salkowski) still doubt the existence of a proper pupil-dilating muscle (*musculus dilatator iridis*). Salkowski also holds that, by reason of the assumed perfect similarity of action of the pupil-dilating and of the vaso-motor branches (see below), these two classes of nerve-fibres must be considered identical,—that the oculo-pupillary and vaso-motor filaments are one and the same. Nevertheless, most authors believe that at least the higher mammalia possess the pupil-dilating muscle referred to, and accordingly also interpret the facts in the way hitherto adopted.

Petit had already observed, with contraction of the pupil after section of the sympathetic, a flattening of the cornea and a dragging of the eye inwards. In 1855 Remak* noticed a raising of the upper eyelid on irritating the cervical sympathetic of animals; and Wagner† and Müller‡, on galvanising the cervical sympathetic of executed criminals,

* "Deutsche Klinik," 1855, No. 27, p. 294.

† "Verhandlungen der Würzburger Phys. Med. Gesellschaft," 1860, Bd. x. p. 11.

‡ *Ibidem*, p. 49.

observed that the eyes were slightly opened. Claude Bernard,* however, was the first who called attention to the fact that contraction of the pupil after division of the sympathetic is constantly accompanied by other symptoms, namely—retraction of the globe of the eye, flattening of the cornea, and decrease in size of the palpebral fissure. These same phenomena, characterised as “*phénomènes oculo-pupillaires*,” he saw also after division of the anterior roots of the two first dorsal nerves, and after section of the spinal marrow at the level of the first and second dorsal vertebræ, that is in the region of the already-mentioned “*centre oculo-pupillaire*.” Irritation of the divided cervical sympathetic, or of the peripheral end of the divided anterior roots, gave the opposite results—prominence of the globe (exophthalmos), bulging of the cornea, and enlargement of the palpebral fissure.

As regards the means by which these appearances are produced, the exophthalmos following irritation of the sympathetic is usually referred to the action of the smooth (unstriped) muscle of the eye (*musculus orbitalis*), discovered by H. Müller,† and situated in the neighbourhood of the *fissura orbitalis inferior*. This muscle—which, though but slightly developed in man, is an important structure in ruminants—has, as Müller has shown, its nerve-supply from the sympathetic system, from the spheno-palatine ganglion; it throws the globe of the eye forwards, and thus acts as the antagonist of the *musculus retractor*, and, in man, also as the antagonist of the *recti muscles*, so that it certainly can have but little power to alter the position of the eye. Besides these, there are still other smooth, unstriped muscles, discovered by Müller,‡ in the upper and lower eyelids of men and the *mammalia*, which may take part in the enlargement of the palpebral fissure and in the protrusion of the globe; and it may be inferred from the above experiments that they also have their nerve-supply from the sympathetic. Finally, Sappey§ has described certain unstriped muscles as existing in the orbital aponeurosis, which co-operate in producing the exophthalmos on irritating the cervical sympathetic.

Some writers also ascribe to the cervical sympathetic

* “*Comptus Rendus*,” 1862, T. lv., p. 382 ff.

† “*Verhandlungen der Würzburger Phys. Med. Gesellschaft*,” 1859. Bd. ix., p. 76. (Sitzung vom 30 October, 1858.)

‡ “*Verhandlungen der Würzburger, Phys. Med. Gesellschaft*,” 1859. Bd. ix., p. 244.

§ “*Sitzung der Pariser Académie des Sciences*,” vom 21 October und 18 November, 1867.

a tonic influence on the voluntary muscles of the eye, and especially several of the fore-mentioned oculo-pupillary phenomena have been connected with this tonic influence. The observation of Petit, that the eye is drawn inwards after division of the cervical sympathetic, is ascribed by Romberg* to disturbance of function of the external rectus muscle—a muscle which has a double motor innervation (from the nervus abducens, and from the ascending branches of the uppermost cervical ganglion), and whose energy is therefore weakened when the tonic force proceeding from the sympathetic is lost.

In the same way Remak† referred the retraction of the upper eyelid in division, and the narrowing of the palpebral fissure in irritation, of the cervical sympathetic to an alteration in the action of the voluntary muscles (the levator palpebrae superioris, retractor plicae semilunaris, and orbicularis palp.), from which he inferred that they are under the control of a tonic power communicated through sympathetic filaments. Schiff also held the same view regarding the oblique muscles, ascribing to them the exophthalmos occurring on peripheral irritation of the divided sympathetic, since, when they are cut through, no such protrusion of the globe takes place. Thus far, according to Schiff, the oblique muscles conduct themselves exactly like organic muscles, as, after cessation of the irritation, the globe of the eye returns slowly to its normal position. Reference will be made later to several other consequences of injury of the sympathetic, such as disturbances of the circulation, of the intra-ocular pressure, of the accommodation, and of the nutrition of the eye (compare “the vasomotor branches of the eye” and “trophic branches.”)

B. *Vasomotor Branches.*—Although Petit mentioned among the results of division of the sympathetic a reddening of the conjunctivæ, the conclusive evidence on this subject originates with Claude Bernard.‡ Division of the cervical sympathetic produces, as he has shown, (in dogs, cats, rabbits and horses), a dilatation of the vessels of the head and neck on the side operated on, and a considerable elevation of temperature, which, measured in the ear, occasionally amounted to 4° to 6° Celsius, and lasted some time. According to Schiff the eleva-

* “Lehrbuch der Nerven Krankheiten,” 2te Auflage ii., 3 Abtheilung, p. 75.

† “Deutsche Klinik,” 1855, No. 27, p. 294.

‡ Bernard: “Comptes Rendus,” T. xxiv., p. 472; “Gaz. Méd. de Paris,” 1852, pp. 75 and 256; “Recherches Expérimentelles sur le Grand Sympathique,” Paris, 1854.

tion of temperature may reach $9^{\circ}\text{C}.$; we, ourselves, sometimes observed differences of $11^{\circ}\text{C}.$ in rabbits directly after division. On the other hand, electrical irritation of the peripheral end of the divided sympathetic causes the previously dilated vessels of the head and neck to contract, and lowers the temperature even to below the normal point. Claude Bernard explained these appearances by paralysis or irritation of the vasomotor nerves of the head distributed in the cervical sympathetic. The rise in the temperature after division is the result of an increased flow of blood to the part through the dilated vessels; while the lowering of the temperature with irritation is the consequence of contraction of the vessels, and therefore of a diminished arterial blood-supply.

X The vasomotor nerves of the head, through the agency of which the vascular-thermic symptoms we are now discussing are produced, have their origin, according to Claude Bernard, in the spinal cord, but not from the same spot as the oculo-pupillary branches. Section of the anterior roots of the two first dorsal nerves gives rise, as we have seen above, to contraction of the pupil, with flattening of the cornea, narrowing of the palpebral fissure, retraction of the globe, but no dilatation of the vessels of the head, no elevation of the temperature of the head; division of the ascending filaments of the thoracic sympathetic between the second and fourth ribs (in dogs), on the other hand, produces only dilatation of the vessels and elevation of the temperature of the same side, whilst the oculo-pupillary phenomena do not appear. Bernard, therefore, came to the conclusion that the centra for the oculo-pupillary and the vascular-thermic filaments of the cervical sympathetic are at different points in the spinal cord, and that the latter branches issue from it at the level of the third and fourth dorsal nerves by means of the communicating branches. This, at least, fixes the lower boundary of that zone of the spinal marrow in which arise those vasomotor nerve-branches which afterwards pass over to the sympathetic nerves. How far upwards this zone continues is in the meantime as undecided as in the case of the oculo-pupillary branches. The opinion formerly held by Budge, that the centre for the vasomotor nerves of the head is situated at the level of the sixth and seventh dorsal vertebræ, must be given up in consequence of the later researches of Salkowski and of Budge himself. Lately certain well-known investigations have placed it beyond doubt that the vasomotor centrum for most parts (in-

cluding the head) is in the medulla oblongata; and, further, not only have Budge's former experiments proved the influence of the pedunculus cerebri on the vessels of the opposite half of the body, but by us also have been defined certain localizable vasomotor districts on the surface of the brain of dogs, in the upper part of the cortical substance of the cerebrum. Thus it is, *à priori*, not improbable that the vasomotor branches distributed in the sympathetic reach their ultimate end in certain spots on the surface of the cerebrum, and this conclusion seems to receive support from a number of observations, by Brown-Séquard and by ourselves, still to be mentioned.

The question has lately risen whether the vasomotor nerves, distributed in the cervical sympathetic, are not, wholly or partly, of a vaso-dilator character. Especially Goltz,* by his researches on the sciatic nerve and lumbar part of the spinal marrow of dogs, has shown that the whole series of phenomena which have hitherto passed for the effects of paralysis, for conditions consequent on a paralysis of vessel-contracting nerves, are produced by a persistent irritation of vessel-dilating nerves. It is obvious that it will completely revolutionize our conception of the functions of these nerves if we regard them as actively-dilating, or, as some authors do, as regulating inhibitory nerves of vessel-contraction—analogues of the inhibitory nerves of the heart. Goltz's results force us to the conclusion that the great peripheral roots, such as the sciatic, by preference or exclusively include such vessel-dilating nerves, and that the corresponding spinal centres really represent centres of vessel-dilatation, not of vessel-contraction. Nevertheless there may be ganglionic centres for vessel-contraction placed in the periphery, in the walls of the vessels themselves, or in their immediate neighbourhood; these may be subject to the influence of the dilating or inhibitory nerves, and may send off excito-motor nerves to the bloodvessels. This latter view seems to find confirmation in the experiments of Putzeys and Tarchanoff,† performed under the direction of Goltz, and also from those of Huizinga‡ on the web of the frog's foot. Those authors, accepting the theory that the vasomotor nerves convey chiefly vaso-dilator impulses, believe themselves also justified in assuming the

* Ueber Gefässerweiternde Nerven. Pfüger's "Archiv. der Gesamten Psychologie," Bd. ix., p. 174.

† "Centralblatt für die Med. Wissensch.," 1874, No. 41.

‡ Pfüger's "Archiv. der Gesamten Physiologie," 1875, xi., p. 207.

longitudinal muscle-cells of the walls of the vessels as the means through which these nerves act, though they have been demonstrated with certainty in only a few, that is, in large arteries.

Here we can touch on this important matter but cursorily, as far only as it stands in immediate relation to our subject. It is true that these investigations, and the conclusions drawn from them by Goltz, referred originally to the vasomotor nerves distributed in the sciatic, or to those of the hinder part of the body in mammalia; but they strongly suggested a transferring of the argument to the cervical sympathetic, and such a transference has already been attempted by Goltz in a very positive way. The above-mentioned fundamental experiment of Bernard, which showed that division of the sympathetic causes dilatation of the vessels of the head, and elevation of the temperature on the injured side, should, according to Goltz, be thus explained—that by means of the incision the vessel-dilating nerves are brought into a state of persistent irritation! He explained in the same way the unilateral increase of perspiration following division of the sympathetic in horses, and the persistent increase of the secretion of the submaxillary gland following section of the cord. Latterly, also, Claude Bernard and Brown-Séquard have been inclined, at least partly, to a similar opinion; the former, inasmuch as he admitted the existence of vaso-dilator along with vaso-constrictor fibres; the latter, as he accepts the appearances following division of the sympathetic, usually regarded as paralytic indications, as the results of irritation.

Such being the state of this question, we regarded it as our duty to repeat the fundamental experiments on the cervical sympathetic, with special reference to the subject in dispute, and in such a way as to ensure complete clearness and certainty. When we examine the older, and often very shortly described, experiments, it is found that, as a rule, too little importance is attached to the determining of the *time* of the occurrence of the vascular-thermic and other phenomena following irritation or division; thus doubts may arise whether, in such a case as lowering of the temperature following irritation of the sympathetic, one has to do with primary phenomena, or rather with secondary phenomena brought on by exhaustion of the irritated nerve. It is clear that if we regard the increase of temperature after section of the sympathetic as the consequence of a persistent irritation,

we should also look for at least a primary raising of the temperature when a divided nerve is stimulated by electricity, not for a lowering of the temperature. Thus, although Goltz at first observed regarding the sciatic that, in opposition to the general opinion, irritation lowers the temperature of the corresponding foot, Putzeys and Tarchanoff soon afterwards stated that this is preceded by a short initial rise in the temperature. The ordinary method of thermometric observation is, in general, not delicate enough to enable us to settle these questions with precision, because the rising and falling of the quicksilver are much too slow to follow with sufficient certainty a rather rapid change in the temperature of the tissues. We have, therefore, both in our experiments on the sciatic (on which we will not enter here, but concerning which we will only state that they proved entirely opposed to those of Goltz, and in favour of the usual, older view), and in our investigations on the cervical sympathetic abandoned the ordinary thermometer, having used exclusively a *thermo-electric* apparatus—a method of observation much more difficult of application, but one which gives more reliable results.

ORIGINAL EXPERIMENTS.—GENERAL PLAN.

Only rabbits were used in the experiments on irritation and division of the cervical sympathetic, as dogs appear to be badly adapted for that purpose from the proximity of the vagus and sympathetic in the neck; besides, the vascular and thin ear of rabbits is a very favourable part for the observation of temperature, and we know that its blood-vessels have their innervation from the cervical part of the sympathetic.

In determining the temperature of the ear we used the electro-galvanometer constructed by G. Meissner and Meyerstein, a detailed description of which cannot be given here.* The superiority of this instrument is the result not only of its *great sensibility*, by means of which even very small differences of temperature are shown with certainty, but also of the *rapidity* with which adjustment takes place. This circumstance enables us to follow even rapid changes of temperature in the tissues with confidence, and to repeat the observations within very short intervals, generally every five seconds.

* See "Zeitschrift für Rationelle Médecin," Bd. xi.

As thermo-electric elements we placed in the circle two of Dutrochet's* needles, which consist of iron and German silver soldered together lengthways as far as the point, according to Poggendorf's proposal. At the upper end of the needles the metals are separated, to admit of being connected, by means of binding screws, with the thick copper wire of the circuit. The needles are covered with a brown varnish, in order that the assumed currents of heat shall not be disturbed by other currents arising from wetting of the dissimilar metals by the fluids of the tissues.

In using the galvanometer we must in the first place ascertain what difference on the scale corresponds to a known difference of temperature in the needle-elements—for instance 1°C . We connected a sensitive thermometer with each of the thermo-needles, placed them in dry test-tubes closed above by wadding stoppers, and put them in water baths of different degrees of heat. The two thermometers then recorded the difference of temperature which simultaneously existed in the two thermo-needles. The scale being now examined, the difference in the expressions found corresponded to the difference of temperature inside the two test-tubes. Thus we find that one degree Celsius in the elements = $13\cdot8$ mmtr. on the scale; or that 1 mmtr. on the scale = $0\cdot0725^{\circ}\text{C}$. We must further notice in what direction the scale is altered when the one or the other element is heated. As a test it is sufficient to bring the hand near one of the two needles, when a deviation in a certain direction immediately follows.

The animal being laid on its back, made fast at the feet with nooses, and at the head by means of Czermak's apparatus for fixing the head, the sympathetic nerve in the neck is laid bare and isolated by means of a glass rod pushed under it. Two platinum wires serve as electrodes. The vivisection-board is set obliquely on a stand, the ears hanging downwards through an opening in the board. The one element is to be thrust longitudinally through the ears of the side under examination, and supported by a cramp-iron applied to the tip of the ear; the other element, fixed by a supporter, is exposed to a constant heat, the point being kept at a stated distance from the steady flame of a petroleum lamp. This element must be brought so near the flame that the latter imparts to it the same amount of heat as the

* See "*Physique Médicale*," Paris, 1855, p. 26.

ear to the other element. This is the case when, on completing the circuit, the index of the scale shows no change, but remains stationary (deducting small oscillations presumed to be caused by the periodic, regular movements in the vessels). The irritation of the nerve was accomplished by means of a secondary current from one of Du Bois Reymond's induction apparatuses.

Our *experiments prove that on irritation in the continuity of the sympathetic and of its peripheral cut end there is an *immediate fall in the temperature, which goes on for some time after the discontinuance of the irritation (20-35 seconds), and is followed by a gradual rise; it does not, however, reach its original point.* The maximum fall amounted to 2.6°C.—on faradisation of the same nerve, repeated after a short interval, 1.1°C. more.

We found exactly the same results, which can only be shortly noticed here, on examination of the sciatic nerve of the dog and rabbit. All these experiments speak in a decisive manner against the doctrine of the existence of vessel-dilating nerves in the trunk of the cervical sympathetic or of the sciatic nerves, as both irritation in the continuity and of the peripheral end, after division, always resulted in a primary lowering of temperature, of varying intensity and duration. Further, these experiments also show that the appearances occurring after the division cannot be ascribed to a persistent irritation caused by the incision; the irritation produced by the section is unimportant, inconstant, and very transitory, manifesting itself in the primary lowering of temperature, whilst the long-lasting and considerable rise in temperature is plainly the consequence of paralysis.

In order to establish more exactly the duration of the rise of temperature, after division of the sympathetic nerve, and the circumstances having an influence thereon, special experiments were instituted in which, as the settlement of the point in question involved repeated comparative observations on both sides of the head, the thermo-electric method of estimating temperature had to be replaced by the ordinary thermometric method.

The earlier experimenters appear to have given but very slight attention to this point, and their conclusions regarding it are both vague and contradictory. Claude Bernard states

* The details of these and of the other experiments mentioned in this Essay are omitted. (Trans.)

that the difference between the two ears is maintained for weeks, or even for an indefinite period. Schiff, on the other hand, observes that it is only when the animals operated upon (dogs, cats, and rabbits) are kept quietly in stalls that the increase of temperature ($5-9^{\circ}\text{C}.$) and dilatation of the vessels are maintained in the ear experimented upon; if the animals are allowed to run about in the open air, and the ears examined when they return heated from their play, breathing quickly, and with the general temperature elevated, the previously warmer ear of the injured side is found to be $1-5^{\circ}\text{C}.$ colder than the other, and its vessels less full. When the animal has rested some time, the former relations of temperature return. The experiments with this end in view, made by us on rabbits, were also at the same time directed to some other points; they proved, in general, that the rise in the temperature of the ear after extirpation of the sympathetic nerve, even when the animals are kept enclosed and at rest, is *not* maintained indefinitely. Rather the difference between the two ears, which might at first amount even to 11° or $12^{\circ}\text{C}.$, became gradually less. In some cases there was not only equality established, but the temperature on the side operated on even sank somewhat below that of the other side, and maintained itself for some time, with certainly not unimportant fluctuations, in this relative position. In one rabbit the flushed and hot condition was passing off only on the eighth day—in another on the second, and more clearly on the following days. Thus the points that presented themselves most prominently for observation were certain relatively small differences between the two sides. If the animals were strongly irritated—as by keeping the mouth and nose closed till dyspnoeal muscular contractions were brought on, or if, by other means, strong movements were produced—not only the temperature on both sides rose, but, *by a further rise in the temperature of the ear operated on, the difference between the two sides was generally increased, rarely somewhat diminished; in the same way, when occasionally the temperature was already high on the side not operated on, the existing difference was usually a little increased by the irritation, rarely influenced in the opposite way.*

Analogous results were obtained by us in a series of experiments on rabbits, in which the cervical sympathetic of one side was extirpated, and the carotid artery of the other side ligatured. Here, also, the temperature on the side on

which the sympathetic was injured was at first appreciably elevated; the difference became gradually less, and eventually the temperature on the other side came to be slightly the higher of the two (by 0.1° — 0.3°C. , on the average)—a change which was noticeable from the sixteenth day.

Extirpation of the sympathetic nerve and ligature of the carotid artery were performed on the same (right) side of a rabbit. In this case the equalising of temperature followed unusually quickly, occurring in a few hours; irritating the animal generally raised the temperature on the uninjured side.

In another rabbit extirpation of the sympathetic and ligature of the carotid were performed on the right side, while on the left the carotid only was tied. Here, too, the striking result was seen, that from the very beginning, immediately after the operation, the temperature of the ear on that side on which there had been division of the sympathetic nerve was, and remained, the lower; the difference was at first but small, but gradually increased, and was most observable when the animal was disturbed.

In connection with this last experiment, it may further be remarked that ligature of the carotid on one side produces, as we have convinced ourselves, a slight lowering of the temperature of the side operated on, amounting on an average to 0.3° — 0.9°C. The fact, therefore, that the temperature of the side on which only the carotid is tied constantly remains higher than that on the side on which the double operation is performed, justifies the conclusion that simultaneous extirpation of the sympathetic prevents the speedy development of the collateral circulation after ligature of the carotid.

With regard to the duration of the oculo-pupillary symptoms, we observed, in a dog in which the united trunks of the vagus and the sympathetic were divided on the left side of the neck, an appreciable contraction of the left pupil four and a half months after the operation.

We may now mention some of the latest and most accurate observations on the *cerebral centra of the cervical sympathetic nerve*, as far as they refer to the oculo-pupillary and vasomotor fibres.

Whilst we were engaged in investigations both on this subject and concerning other vasomotor cerebral centra, Brown-Séguard* published some experiments on rabbits and

* Production des effets de la paralysie du nerf grand sympathique cervical par l'excitation de la surface du cerveau: "Archiv. de Phys.," 2 ser., T. ii., No. 6; Oct. till Decr. 1875, page 854.

dogs, according to which *irritation of the surface of the brain produced the appearances of paralysis of the sympathetic of the same side*. In the first place it is to be observed that he effected the supposed "excitation" by scorching the surface of the brain with a hot wire; he regards this proceeding as a thermic irritation of the surface of the brain, whereas it plainly acts as a destructive and paralysing agent, at least on the parts of the brain surface most immediately concerned, as we have proved in a large number of experiments, both on the motor regions of the surface of the cerebrum of dogs and on the vasomotor centra of the extremities—the existence of which centra was also demonstrated by us. Further, some of Brown-Séquard's stated results are very indefinite and variable, and some of them very questionable, at least in their explanation. He states that, in his experiments, among the appearances that may be regarded as corresponding to those of division of the sympathetic he observed only *one* that was constant, *the narrowing of the palpebral fissure*, and that only when the injury was done to the right side; on the left side the effects of the burning, as he adds in a note, took a very different form. The other oculo-pupillary and vasomotor phenomena (contraction of the pupil, injection of the conjunctiva, elevation of the temperature of the ear) were more inconstant and less marked than after division of the sympathetic. As regards specially the unilateral elevation of temperature, Brown-Séquard merely remarks that generally the ear on the side operated on is somewhat warmer; the only experiments recorded by him prove differences of only 0.1° and 0.2°C. , on which no great value can be set, at least compared with the important results of division of the sympathetic. Irritation of the middle lobe of the brain produces the appearances most intensely, and more so when stimulated in its median than in its lateral parts; the posterior lobe responds more feebly, and the anterior most feebly of all.

But these phenomena are also sometimes seen, temporarily, in lesions of the scalp, the pericranium, and the meninges; and from that Brown-Séquard infers that he has to do with a reflex action caused by irritation of the sensitive terminations of the trigeminus distributed in the investing structures of the brain. Although our attitude with regard to this question has always been quite different to that of Brown-Séquard—since, after the discovery of vasomotor centres in the cortical substance for the extremities of the opposite

side, we also assumed that there should be analogous local centres, in the neighbourhood of those just mentioned, for the vasomotor nerves of the head distributed with the cervical sympathetic—we thought Brown-Séguard's statements should, in the first place, undergo a rigid examination. We found, however, that their exactness was confirmed to only a very slight extent. It must first be denied that the burning, or (as Brown-Séguard expresses himself) the "thermic excitation," of the surface of the brain is uniformly followed by a contraction of the palpebral fissure on the injured side. One is liable to err in this matter, as the contraction of the skin following a wound in the neighbourhood of the orbit may easily be taken for a slight narrowing of the palpebral fissure. A constant dragging forward of the palpebra tertia in rabbits was seldom noticed by us. In certain cases, on the other hand, we observed, especially after burning in the region of the middle (parietal) part of the almost unconvoluted convex surface of the brain in rabbits, an inequality of the pupils and of the palpebral fissure, and even in the prominence of the globe of the eye on both sides. After such thermic excitation, sometimes immediately, and sometimes a few hours later, the right pupil appeared decidedly more contracted (though still responding to light), the palpebral fissure smaller, and the globe of the eye less prominent than on the left—the uninjured—side. These phenomena certainly admit of a double explanation, especially as they appeared in only a very slight degree; the cause of the unsymmetrical action might as justly be assumed to be an irritation of the unwounded side as a paralysis of the injured side.

In dogs, notwithstanding our exceedingly numerous experiments, we have not observed similar appearances; neither the disturbance of larger parts of the brain-surface by heat, nor the electric or chemical irritation of the same, was followed by decided oculo-pupillary phenomena on the same or the opposite side. As regards the cerebral centrum of the vasomotor fibres of the sympathetic, we have not yet arrived at decided results, notwithstanding the large number of our experiments on rabbits and dogs. The easily-performed experiments on rabbits showed, in partial, unilateral disturbance of the surface of the cerebrum by an iron at a red heat, either no difference, or such a small and variable difference, in the temperature of the ears, that no positive demonstrative value can be attached to them. On

the whole, we noticed, contrary to Brown-Séquard's observations, much more frequently an elevation of the temperature of the ear on the uninjured side; this amounted sometimes to $0.7^{\circ}\text{C}.$, and in one case it rose, four hours after the operation, to $1.7^{\circ}\text{C}.$ It is also noteworthy that in the rabbits operated on for this purpose, after irritation of large parts of the surface of the brain, rotatory movements usually occurred towards the side opposite to that injured.

We will not here refer further to the probable causes of these phenomena, but will only remark that they obviously do not arise from affection of the basilar part of the brain (the pons, pedunculus cerebri, &c.), since, as was proved by autopsy, the thermic irritation reached only to a depth of 1—1.5 mmr. In electric irritation of the exposed brain-surface of the rabbit, and especially in the lateral part of the posterior half of the brain external to the sulcus which runs parallel and near to the superior longitudinal fissure, we obtained, in four experiments, a slight but decided and almost instantaneous fall in the temperature of the opposite ear, amounting to 0.2 — $0.4^{\circ}\text{C}.$; on the irritated side itself there was either a slight lowering of temperature (0.1 — $0.15^{\circ}\text{C}.$) or no change. After the irritation was ended, the temperature rose again quickly to its former level, so that both sides were equal. Although the constancy of this result certainly astonished us, we would not, on account of the smallness of the differences, attribute to them very great importance.

The experiments on dogs gave a really negative result. Disturbance, or electrical or chemical irritation, of those parts of the cortical substance (especially of the parts of the gyrus post-frontalis lying behind the sulcus cruciatus) which, according to our showing, influence the temperature of the extremities of the opposite side, and disturbance or irritation of those other superficial portions of the cortical brain-substance lying more anteriorly, posteriorly, or externally, leaves the temperature of the ear on both sides equal, or at least nearly unchanged.

VASOMOTOR BRANCHES OF SPECIAL ORGANS.

1. *Vasomotor Branches of the Eye.*—*Intraocular Pressure.*—The experimental investigation of the functions of the vasomotor nerves of the eye which proceed from the cervical sympathetic, and of their influence on intraocular pressure

(which is of special importance in the pathogeny of glaucoma simplex), cannot yet be said to be complete, the results hitherto obtained being unfortunately somewhat contradictory.

That the conjunctival vessels, at least in part, had their nerve-supply from the sympathetic, was known long ago to both Pourfour du Petit and Claude Bernard, who affirmed that division of the cervical sympathetic excited congestion of the conjunctiva. As regards the vessels of the interior of the eye, Wegner* first found (in rabbits) that division of the sympathetic caused dilatation of the vessels of the iris on the same side, while irritation of its peripheral end was followed by contraction. Division of the trigeminus, indeed, also produced dilatation of the vessels of the iris, but, as Wegner holds, only because the branches coming from the sympathetic, which lie on the inner side of the trigeminus within the cranium, were cut at the same time. The choroidal and retinal vessels also, as ophthalmoscopic investigation shows, are influenced by the sympathetic in the same way. *Division of the sympathetic is attended first by a gradual diminution of the intraocular pressure*, amounting to 4—8 mmtr. (measured by the manometer in the anterior chamber of the eye), which Wegner is disposed to ascribe to dilatation and paralysis of the bloodvessels. In two out of four experiments irritation of the sympathetic produced a slight increase of tension which soon passed off again.

Adamück† observed, after dividing the sympathetic of cats under chloroform, a diminution of the intraocular pressure of 1—2 mmtr.; this was sometimes persistent, sometimes followed by a secondary increase of tension. Irritation of the cranial end of the divided sympathetic was always followed by a slow increase of tension of 2—4 mmtr.; after remaining some time at this level it began to sink, the decrease continuing after cessation of the irritation, and gradually came back to the normal point. The lowering of the pressure appears at the same time as the dilatation of the pupil and the protrusion of the eyeball, and is brought on by diminution in the blood-supply, as in ligature of the carotid. The preliminary increase of tension probably has its origin, according to Adamück, in the accommodation apparatus, at least the experiments on eyes under the influence of atropine speak for that view. If the ciliary muscle of cats be, as far

* "Archiv. für Ophthalmologie," xii., 2, pag. 1, 1866.

† "Centralblatt für die Med. Wissensch.," 1866, pag. 561; 1867, pag. 433.
"Annales d'Oculistique," lviii., p. 5.

as possible, paralysed by atropine, the pressure within the eye under the influence of atropine is 2 mmtr. less than in the other: thus, division of the sympathetic results, not in diminution, but in immediate increase, of the intraocular pressure; while irritation of the cranial end is followed by decrease, not by increase.

Irritation of the cervical sympathetic sets two apparatuses in action, which, in relation to the intraocular pressure, so oppose each other, that, according as the energy of the one or of the other preponderates, increase or diminution of the pressure follows. The one apparatus, the vasomotor, lessens the pressure; the second, situated apparently in the internal muscles of the eye (in the accommodation apparatus), augments the pressure.

Grünhagen* confirmed part of Adamück's conclusions, but nevertheless endeavoured to explain the primary increase of tension in irritation of the sympathetic by the action of the external muscles of the eye, especially by that of the orbital muscle of Müller. In opposition to this idea, Adamück holds that the cause of the increase of tension can be found only in the contraction of the internal muscular fibres of the eye, especially of Müller's muscles of the choroid, or perhaps of a part of the ciliary muscle. These muscles, probably having their nerve-supply from the sympathetic, are said to draw forward the lens, and thereby to increase the tension in the anterior chamber. Those filaments of the sympathetic whose action is to increase the pressure, do not, according to Adamück, pass through the ganglion ciliare, and in the orbit also they do not lie close to the pupil-dilating fibres, but rather to those of the optic nerve.

Hippel and Grünhagen have come to the conclusion that while those nerve-fibres through which the iris may be made to contract enter the cervical sympathetic about the middle part of its course, most of the vessel-contracting branches of the eye join it at the level of the ganglion cervicale supremum. Thus, when the middle part of the cervical sympathetic is stimulated the majority of the vessels of the eye remain unaffected. The increase of the intraocular pressure, which occurs simultaneously (in cats and dogs), depends, according to Hippel and Grünhagen, on contraction of the unstriated muscles of the orbit, which compress the globe of the eye and hinder the return of the venous blood. On

* "*Zeitschrift für Rationelle Medicin.*," 1866, Bd. xxviii., p. 238.

the other hand, irritation of the ganglion supremum alone produces always a decrease of the intraocular pressure in cats and rabbits—whilst extirpation of this ganglion is followed by an increase. Hippel and Grünhagen further ascribe to the trigeminus an influence on the intraocular pressure, inasmuch as they believe that in it vessel-dilating nerves of the eye are distributed.

The influence of the ganglion supremum was also thus far confirmed by Sinitzin,* that he, after extirpation of this ganglion, always observed injection of the vessels of the fundus oculi on the side operated on. On ophthalmoscopic examination it was found that the choroidal vessels had increased in volume, their anastomoses were more distinct, and the whole fundus of the eye appeared decidedly redder than on the other side. The temperature of the eye was also raised; in the conjunctival sac, and under the capsule of Tenon, the difference amounted to 0.9 to 2.4° C. in favour of the eye operated on.

2. *Vasomotor Branches of the Brain and of its Membranes. Intracranial Pressure.*—The exact relations of the cervical sympathetic to the innervation of the cerebral vessels, and its influence on the intracranial pressure, have not yet been made out with any degree of certainty.

Descriptive anatomy teaches that the numerous nerves found in the pia mater, which follow the vessels in a plexiform arrangement and enter partly with them into the cortical substance, rise, at least partially, in the sympathetic plexus vertebralis, while others certainly come from the efferent cranial nerves, especially the trigeminus. That the sympathetic exercises some control over the cranial vessels is thus at least not improbable. Donders and Callenfeld† noticed contraction of the cranial vessels on irritating the cervical sympathetic. Nothnagel,‡ also, was convinced, through several of his division experiments, that the cervical sympathetic, and especially the ganglion supremum, have a share in the innervation of the vessels of the pia mater. Powerful electrical irritation of the nerves of sensation of the skin produced, in rabbits, a reflex contraction of the arteries of the pia mater; the same occurred also after division of the sympathetic between the superior and middle cervical ganglia, and less markedly after extirpation of the superior ganglion,

* "Centralblatt für die Med., Wissensch.," 1871, No. 11.

† "Meissner's Jahresbericht," 1856, p. 348.

‡ "Virchow's Archiv.," Bd. 40, p. 203.

being then discernible only by means of a magnifying glass. Other investigators (Schultz,* Riegel and Jolly†), have nevertheless either quite denied, or at least admitted only in a very modified way, that the sympathetic exercises such a function. Riegel and Jolly found, in division and extirpation experiments, that neither the trunk of the cervical sympathetic nor the ganglion supremum can be said always to include vasomotor branches for the vessels of the pia mater.

Lately Fischer‡ has submitted the action of the sympathetic on the blood-pressure in the arteries of the head, and on the circulation in the brain and its membranes, to an experimental examination. The cervical sympathetic was irritated sometimes by means of the induced current, sometimes by the constant current. Experiments on horses, with Ludwig's hæmato-dynamometer, showed, with faradisation, a pretty regularly occurring increase of pressure in the arteria maxillaris externa, together with increased tension of the arterial walls; irritation with the constant current gave no obvious result. The investigations on the subject of the influence of the cervical sympathetic on the intracranial pressure were carried on in cats, the brain-pressure being taken with the kymographion, through an opening in the dura mater. Faradic irritation of the isolated sympathetic increased the brain-pressure in five cases out of eight; in three cases there was a trifling decrease; the constant current produced either no result, or a very trifling increase of pressure when the circuit was closed. The simultaneous faradisation of both sympathetic nerves was followed, in four experiments, by a preliminary quick increase of brain-pressure, succeeded by a decrease when the irritation was continued; in all four cases convulsions occurred, in the form of clonic extensor-spasms and opisthotonos, which were never observed in unilateral irritation, and which were probably caused by the decrease or cutting off of the arterial blood-supply to the brain.

3. *Vasomotor Branches of the Ear.*—The vessels of the cavity of the tympanum become dilated, as Prussak§ has proved, after division of the cervical sympathetic of the same side. The inference is thus easy, that the variations in the intra-auricular pressure (pressure within the labyrinth) are caused by much the same conditions as those of the intraocular pressure.

* "Petersburger Med. Zeitschrift," 1866, xi., p. 122.

† "Virchow's Archiv.," 1871, Bd. 52, p. 218.

‡ "Deutsches Archiv. für Klinische Medicin.," Bd. xvii., Heft i., 1875.

§ "Meissner's Jahresbericht," 1868, p. 440.

C. *Trophic Branches*.—It is exceedingly probable that the cervical sympathetic, in many ways, exerts an influence on the glandular secretions and the nutrition of the head. Whether this proceeds, entirely or partly, from vasomotor fibres, or whether it is to be regarded as quite independent of these, and as the function of special secretory or trophic nerve-fibres, cannot yet be decided with certainty by means of the facts before us. We will, therefore, only shortly notice the most important observations having a bearing on the point.

1. *Salivary Glands* (Glandula submaxillaris, sublingualis, parotis).—In the course of the sympathetic the glands just named receive nerves, irritation of which provokes a secretion, scanty, but rich in its specific elements, and therefore tenacious and often gelatinous.

Since irritation of the sympathetic nerves, as Claude Bernard* first observed, excites contraction of the vessels of the glands, retards the circulation through them, and gives a darker appearance to the venous blood, it might seem most reasonable to unite the vasomotor with the secretion-controlling function, and to trace back the latter to changes in the filtration-pressure in the glandular capillaries, and to the more or less abundant supply of oxygen. Various physiological facts, however, appear to be antagonistic to this view: the secretion caused by irritating the nerves can be produced in glands through which there is absolutely no circulation; and when the discharge of the secretion occasioned by nerve-irritation is artificially impeded the pressure in the excretory duct of the gland may be greater than that in the supplying arteries (Ludwig). The theory that there are special secretory branches for the salivary glands is further supported by Pflüger's discovery that certain nerve-fibres terminate directly in the cells of the glands. This refers both to the glandular nerves distributed in the sympathetic, and to those in the facial and trigeminal nerves; on irritation of the latter, as is well-known, a specifically different, copious, and thin secretion is poured out. In paralysis of the sympathetic branches by curare, and after extirpation of the ganglion submaxillare, there occurs, as Bernard has shown, a continuous (paralytic) secretion, which may be increased by irritation of the organs of taste, but which, on the occurrence of structural alteration of the gland, speedily abates. It has been inferred therefrom

* "Liquides de l'organisme," 1859, f. vi., p. 300; "Comptes Rendus," 1862, ii., p. 343, and "Journal de l'Anat. et de la Phys.," 1864, p. 311.

(Bernard) that the chorda tympani includes certain filaments whose function is to preside over secretion in the salivary glands. It has been proved also by Heidenhain that stimulation of the nerves of secretion, especially of those cerebro-spinal glandular nerves which excite a very fluid secretion, leads to a change of the protoplasmic cells into mucus-cells (by mucus-metamorphosis of the cell contents), and to further division of the newly-formed cells; facts which favour the view that there are special nerves of secretion.

2. *Nasal Mucous Membrane.*—Whether the secretion of the nasal mucous membrane is influenced by the sympathetic nerve, is still doubtful. Prevost,* by electrical irritation of the ganglion nasale, produced a congested state of the membrane; stimulation of the divided sympathetic, however, had not the same result. Vulpian,† also, found that irritation of the sphenopalatine ganglion was followed by increased secretion on the corresponding side of the nose.

3. *Lachrymal Gland.*—Concerning the influence of the sympathetic on the secretion of tears, Herzenstein‡ and Wolferz,§ after carefully watching the effects of irritation, could formulate no definite conclusion; nevertheless, the greater number of Wolferz's experiments would seem to indicate that the sympathetic does exercise some control over the secretion—a theory with which Demtschenko's|| results agree.

The principal nerves of secretion of this gland are derived, however, from the trigeminus, in the N. lacrymalis and N. subcutaneus malæ.

4. *Nutrition of the Eye.*—Valentin,¶ Reid,** Volkmann,†† and others, state that after division of the sympathetic there are changes in the eye analogous to those occurring after division of the trigeminus. Brown-Séquard also has observed, after section of the sympathetic in guinea-pigs and rabbits, a gradual atrophy of the eye of the side concerned. It is generally admitted that this is due chiefly to paralysis of vasomotor or trophic nerve-fibres which join the trigeminus, and are found in the gasserian ganglion and its first division.

* "Meissner's Jahresbericht," 1868, p. 327.

† "Archiv. de Phys. Normale et Pathologique," 1869.

‡ "Beiträge zur Physiologie und Pathologie der Thränenorgane." Berlin, 1868.

§ "Experimentelle Untersuchungen über die Innervationsmenge der Thränendrüse," diss. Dorpat., 1871.

|| "Archiv. für die Gesamte Physiologie," Bd. vi., p. 191.

¶ "Funct. Nerv.," p. 109.

** "Phys. Anatom. and Path. Researches," Edinb., 1848, p. 296.

†† R. Wagner's "Handwörterbuch," ii., p. 621.

Others hold that the so-called ophthalmia neuroparalytica, following section of the trigeminus, is generally but the consequence of the want of protection of the eye against mechanical and other injuries, the result of the anæsthesia of the cornea. We should state, further, that in dogs, in which the united vagus and sympathetic were cut, we could not find, 4½ months afterwards, any trace of ophthalmia or atrophy of the eye.

Sinitzin* has lately attributed to the sympathetic a function almost directly opposed to that mentioned by the above-named authors. He found that after extirpation of the superior cervical ganglion, the cornea of the same side, in comparison with the other, offered considerably more resistance to foreign bodies; while these (particles of glass, &c), on the sound side, produced more or less violent conjunctivitis, pannus, purulent infiltration of the cornea, with ulceration and loss of tissue in the neighbourhood of the infiltration, or violent iritis, and panophthalmitis—the side operated on showed almost none of these conditions. *The neuro-paralytic phenomena in the eye did not appear after division of the trigeminus in the cranium (immediately in front of the gasserian ganglion), if, not long before the operation, or immediately thereafter, the superior cervical ganglion was extirpated.* Even the neuro-paralytic phenomena arising from primary division of the trigeminus—when they had not made too much progress, the cornea being still moist and clear—disappeared entirely in the course of 2—4 days on tearing out the ganglion; even when the morbid changes had become too extensive to permit of perfect recovery, extirpation arrested their progress, and produced a certain amount of improvement. The same thing occurs with the ulcerations on the lips and eyelids after division of the trigeminus. Not long ago Eckhard and Senftleben† repeated these experiments, with results not entirely confirmatory of Sinitzin's statements.

5. *Nutrition of the Brain.*—Brown-Séquard‡ states that within a few months after division of the cervical sympathetic in guinea-pigs and rabbits, he observed atrophy of the corresponding half of the brain. After him Vulpian obtained the same results.

D. *Cardiac Excitomotor Branches.*—In the cervical sympathetic are included certain nerve-fibres which are generally sup-

* "Centralblatt für die Med. Wissensch.," 1871, No. 11.

† "Virchow's Archiv.," 1875, 65^{ter.}, Band. p. 69.

‡ "Archiv. de Phys.," 2 Sér., t. ii., No. 6, Octr. bis Dec., 1875, p. 854.

posed to convey influences which *accelerate the heart's action*. These are the cardiac sympathetic branches rising from the cervical ganglia—the N. cardiacus superior from the upper ganglion, the N. cardiacus medius from the middle, and the N. cardiacus inferior from the lower ganglion. All these nerves communicate, by many anastomoses, with the corresponding spinal cervical nerves and with the hypoglossal and vagus. Besides these (according to V. Bezold*) there are certain stimulating cardiac fibres which, arising in the brain, pass through the cervical and upper part of the dorsal portions of the spinal cord to the ganglion cervicale inferius and the superior thoracic ganglion, to be distributed eventually in the tissues of the heart. The belief in such an excito-motor cardiac nerve-system, according to V. Bezold's meaning, stimulating directly from the brain, has been very much debated; against it it has been specially objected that the presumed excitatory influence on the motions of the heart is to be referred entirely, or for the most part, to the vasomotor fibres in the sympathetic, or to the reflex action of the N. depressor (Ludwig and Thiry,† Traube‡, M. and E. Cyon;)§ and V. Bezold|| himself has admitted that the vasomotor fibres co-operate in effecting the changes in the heart's action and in the blood-pressure.

Lepine,¶ with Bochefontaine and Tridon, have lately found that irritation of certain spots on the surface of the brain of dogs (gyrus postfrontalis and a part of the gyrus præfrontalis) with a weak induction current gave rise to not only a considerable increase of the pressure of the blood in the crural artery, equal to 7 cm. mercury, but also at the same time to an *acceleration of the heart's beat*. With the vagus nerves intact, and a powerful current, there was, on the other hand, a decrease in the number of the heart's contractions. It thus appears not impossible that there may be, in that situation, a cerebral centrum for the cardiac accelerating branches of the sympathetic nerve, as V. Bezold originally supposed; while it is just as possible, and, according to our formerly mentioned

* "Untersuchungen über die Innervation des Herzens." Leipzig, 1863.

† "Centralblatt für die Med. Wissensch.," 1866, No. 32.

‡ "Wiener Sitzungsberichte," xlix., ii., p. 421.

§ "Berliner Klinische Wochenschrift," 1866, No. 51.

|| "Centralblatt für die Med. Wissensch.," 1866, No. 51. "Reichert's u. du Bois Reymond's Archiv," 1867, p. 398.

¶ "Centralblatt für die Med. Wissensch.," 1867, No. 2 and 23.

¶ Recherches sur les centres moteurs de l'encephale. "Gaz. Med.," 1875, No. 25.

investigations, even more probable that we have to do rather with simple vasomotor centres situated in the forementioned spots in the cortical brain substance, and that the acceleration of the heart's action following irritation of the centres must be regarded only as the result of the increase of the arterial pressure. Thus, the existence of a cerebral centre for the cardiac excitomotor branches remains, in the meantime, just as uncertain as that of a centre for the oculo-pupillary branches, or for the excitomotor branches of the vessels of the head.

The various inquiries as to the effect of irritation of the cervical sympathetic (especially of its lower part and of the superior and middle ganglia) on the heart, have given but unsatisfactory results; it is only established that, through the ganglion stellatum and the upper thoracic ganglion connected with it, the heart may be made to act more rapidly, or may be set in motion again after having come to a standstill. Cl. Bernard, V. Bezold, M. and E. Cyon, and Schmiedeberg, are all agreed on that point. When considering the functions of the thoracic and abdominal parts of the sympathetic system we will again refer to the possible explanation of these facts, and some others connected with them. Here, in conclusion, it may be mentioned that some authors believe also in the existence of so-called "pressor" nerves in the cervical sympathetic and in the vagus (especially in the ramus laryngeus superior), irritation of which increases tone reflexively, and which thus act in opposition to the ramus depressor of the vagus discovered by E. Cyon (Aubert and Roever). Concerning the origin and course of these branches comparatively little is known; they may, however, be identical with those nerves which tranquilize reflexively the irritation of the vagus.

II. *Experimental Observations on the Functions of the Cervical Sympathetic Nerve in Man.*—The present observations can naturally refer only to irritation experiments, which were principally conducted on living men by means of percutaneous faradisation or galvanizing of the cervical sympathetic.

Besides these we have some observations on direct irritation of the exposed cervical sympathetic in executed criminals. Fragmentary as these are, they support the view that its functions in men coincide in reality with those discovered in other mammalia. This refers specially to the oculo-pupillary branches, since, as was already mentioned, R. Wagner and H. Müller observed the eyelids opening on electrical irritation

of the cervical sympathetic of executed criminals. With regard to the cardiac excitomotor branches, an observation of Henle's* is of interest; he noticed that, in the case of a man who was beheaded, the movements of the auricle ceased in about 25 minutes after death, and that on applying the wires of an induction apparatus to the peripheral end of the divided sympathetic on the left side, the rhythmic contractions began again almost immediately. Nevertheless, Henle attached no great importance to these facts, because repeatedly, after prolonged pauses, the auricle began again spontaneously to beat.

As regards percutaneous irritation of the cervical sympathetic, Gerhardt† obtained, in one case, a very slight dilatation of the pupil by faradisation and galvanisation, applying the cathode between the inferior angle of the maxilla and the sternocleidomastoid muscle, and the anode to the arch of the palate on the same side. In one case, in which there was a swelling on the right side of the throat, in front of the clavicle, Gerhardt could, by compressing this swelling with his fingers, produce a dilatation of the pupil and a decided lowering of the frequency of the pulse, the latter, most likely, through simultaneous mechanical irritation of the vagus.‡

We have performed many experiments to show the *influence of percutaneous galvanisation on the pupil, the frequency of the pulse and the blood-pressure* in men, the principal results of which are stated below. In the first place let it be understood that for the point of irritation for the ganglion cervicale supremum (according to Remak) we took the so-called fossa auriculo-mastoidea, for that of the ganglion cervicale medium the neighbourhood of the transverse processes of the 4th and 5th cervical vertebræ, and for that of the ganglion cervicale inferius a point near the transverse processes of the two last cervical vertebræ. That a current of electricity may pass through the cervical sympathetic in galvanising these spots is shown by the researches of Burckhardt§ and Ziemssen|| on the dead body, inasmuch as they, by means of insulated

* Handbuch der Nervenlehre. Braunschweig, 1871, p. 575.

† "Zur Casuistik der Gehirn Krankheiten, Jena'sche Zeitschrift für Medicin und Naturwissenschaft," 1864, I., p. 200.

‡ See "Czermak, Prager Vierteljahresschrift," Bd. 100, p. 30, 1868. "Rossbach, über mechanische vagus-und sympathicus-reizung bei Mediastinaltumoren," Dissert. Jena, 1869.

§ "Ueber die polare Methode, Deutsches Archiv. für Klinische Medicin," 1878, Bd. viii, p. 100.

|| "Die Electricität in der Medicin," 4te Auflage, Berlin, 1872, p. 38.

conducting needles thrust into the sympathetic from behind, and connected at their free ends with the galvanometer, succeeded in proving the transmission of a weak current through the nerve when the external electrodes were placed on the angle of the lower jaw and on the manubrium sterni.

Original Experiments.—Our experiments on healthy persons gave the following results:—

When the anode of a constant battery of 20—40 of Siemens' elements is placed on the manubrium sterni, and the cathode immediately behind and below the angle of the jaw, a slight dilatation of the pupil on the irritated side instantaneously occurs on closing the circuit, followed by a gradually increasing contraction when the current is kept up. Sometimes these phenomena do not appear on the first closure of the circuit, but they are produced distinctly when the current, after being continued for some time ($\frac{1}{4}$ — $\frac{1}{2}$ minute), is interrupted, and again closed; and the same result follows each successive closure. Generally the momentary dilatation corresponding to the completion of the circuit is so slight as to escape direct objective observation; but one may bring it subjectively into view by aid of the Pupilloscope* of Giraud-Teulon, first described by Houdin. The construction of this instrument was suggested by the fact that there are on the retina a number of dispersion-circles whose size and relative distance are correspondingly modified by the slightest change in the diameter of the pupil. At the instant that the electric connections are completed, the dispersion-circles are seen by those qualified for personal observation to be suddenly enlarged; but while the current continues they gradually become smaller. The effect of interrupting the current is very variable; sometimes increase, frequently decrease in size, and occasionally no appreciable change is observed in the dispersion-circles. In some cases, when employing a very powerful current, which is kept up for some time, a persistent and objectively demonstrable dilatation of the pupil occurs, which continues even after withdrawal of the current.† As regards the causes of these phenomena, it may clearly be inferred that the momentary dilatation of the pupil occurring on closing the circuit is due to a convulsive

* See Zehender, "Klinische Monatsblätter für Augenheilkunde," Sept. 1867, p. 276.

† Those patients in whom such proceedings cause giddiness and stupor, and various phenomena in the organs of the senses (Photopsia, &c.), are evidently not suitable subjects for experiment.

action in the sympathetic pupillary branches; and that the inconstant dilatation on interrupting the current, and that which more rarely occurs while the current is kept up, correspond to the galvanotomic stimulation of other motor nerves. The probability of this explanation is increased by the following considerations:—(a) That the results of reversing the current (the anode being behind the angle of the jaw) are much more uncertain; (b), that they generally disappear when the negative electrode is removed from the spot corresponding to the ganglion supremum and placed, for instance, in the neck, on the lower cervical spinous processes; (c), that when the electrodes are applied symmetrically behind and below the angle of the jaw, these phenomena, in the eye corresponding to the cathode, are more marked and constant in their occurrence. Nevertheless, there always remains the objection that the dilatation of the pupil in percutaneous galvanisation originates, not from direct stimulation of the sympathetic, but reflexively from the irritated nerves of sensation in the skin. Claude Bernard has shown (and we found it confirmed by our experiments on animals and men) that a powerful irritation of sensory nerves may produce reflex dilatation of the pupil. In the same way, and oftener on powerfully stimulating the crural nerve with an electric current, we have obtained the same result.

After the prolonged operation of a strong, continuous current, flowing in the above-mentioned direction, we frequently observed, when the circuit was still complete, a fall in the frequency of the pulse, amounting to 4–16 beats per minute in those cases in which the normal frequency was 60–80; and with this was conjoined a decrease, discoverable by palpation, of tension and pressure in the carotid of both the irritated and the non-irritated sides, and even in the radial arteries. The information supplied by Marey's sphygmograph was more exact, especially with reference to the carotid. The typical appearance of the previously normal carotid tracings was strikingly altered, when the galvanic current was passed in the manner before described. The line of ascent becomes more oblique, and deviates towards the right, while the pointed apex almost or entirely disappears. The descending curve which should follow the primary ascending line becomes a broad plateau, 2 mmtr. in length, sometimes horizontal, and sometimes presenting another more or less steep ascent. The end of this portion of the curve, which is at the same time the beginning of the actual

line of descent, is formed by the summit of the first secondary wave, which thus rises to the same height as, or even higher than, the primary wave. The line of descent falls somewhat gradually, and the last (great) indentation in the tracing, with its corresponding line of ascent, is flat or rounded.

These changes are sometimes clearly seen, especially the more oblique direction of the line of ascent, in the radial tracing. On the other hand, there is wanting here the flattening or secondary elevation at the apex, so characteristic of the carotid curve; the apex is more pointed than under normal conditions, and the first secondary wave appears clearly developed in the course of the line of descent. Thus, from these statements regarding the carotid and radial curves, partly corroborative and partly contradictory, it may be conjectured that in the before-mentioned method of galvanisation we have to do with two factors, one acting generally, the other locally. Concerning the first factor there can be no doubt; it is the retarding and the weakening of the heart's action, which show themselves in the decrease in the frequency and tension of the pulse, and graphically in the diminished height and more oblique direction of the line of ascent. The second local force is perhaps to be sought in the influence of the electric current on those vasomotor nerve-fibres of the head distributed in the cervical sympathetic, whereby the arterial tone in the region supplied by the carotid is diminished, and the blood-pressure in the carotid itself considerably lowered. Very specially in favour of this view is the circumstance that when a strong current is sent in a downward direction through the neck, from the vertebral column to the brachial plexus, the same pulse-tracings are obtained (while the circuit is closed) in the radial artery of the irritated side as in the case of the carotid just mentioned. By this method also, as in various other ways in which galvanisation is practised (such as the transmission of a powerful descending current longitudinally in the vertebral column, &c.), a transitory diminution in the frequency of the pulse occurs. We believe that this is caused by a reflex stimulation of the inhibitory nerve of the heart, the vagus; at all events it can scarcely bear any relation to the sympathetic system. X

Experiments lately performed by us, using the thermoelectric apparatus formerly described, proved clearly that electrification of the neighbourhood of the cervical sympathetic had a decided effect on the temperature of the correspond-

ing side of the head. When a strong faradic current was passed the temperature of the face, during the continuance of the irritation, sank 0.5 to 0.7°C. When the irritation was effected by means of a constant current, the cathode being in the auriculo-mastoid fossa, there was a momentary lowering of temperature on the same side of the face, whilst with the anode in the same position there was almost no result.

These experiments not yet being concluded, we abstain from giving a more complete account of them.

Rockwell and Beard* have prosecuted some inquiries on living men, concerning the *influence of percutaneous galvanisation of the cervical sympathetic on the vessels of the fundus of the eye*. A current of 10—25 elements was used for 2—5 minutes, the anode being placed in the auriculo-mastoid fossa, and the cathode on the manubrium sterni, or at the side of the sixth cervical vertebra. The results, which coincided exactly with those of our own experiments, were sleepiness, a variable sensation of warmth, changes in the pupil, and lowering of the rate of the pulse. As regards the ophthalmoscopic appearance of the vessels of the eye, three practised observers give quite contradictory statements. One of these† noticed Hyperæmia, followed by Anæmia, of the retinal veins; a second,‡ loading of the retinal veins; a third (Hackley), only a slight contraction of the arteries. We shall not be surprised at the relative unsuccessfulness of these experiments when we consider that, according to Grünhagen and Hippel, probably only the vasomotor nerves of the iris, and not those of the choroid and retina, are supplied by the cervical sympathetic, as was formerly mentioned.

In conclusion, we may here notice *the changes in the secretion of perspiration on galvanising the cervical sympathetic*.

In several cases M. Meyer§ made the important observation that by galvanisation of the cervical sympathetic the secretion of perspiration in the arm is increased. Thus, when the cathode was placed on the ganglion cervicale supremum, and the anode on the transverse process of the seventh cervical vertebra of the opposite side, there was a rise in the temperature of the arm on the side corresponding to the

* "On Medical and Surgical Electricity," 1871.

† Roosa.

‡ Loring.

§ "Berliner klinische Wochenschrift," 1868, No. 23; 1870, No. 22.

cathode, and the perspiration appeared in drops in the palm and on the finger-tips. In our opinion this result is to be ascribed only to currents which, in using the electrical apparatus in the way described, enter the brachial plexus or spinal marrow; we believe that most probably it has no connection with the sympathetic. Further, in a case of ephidrosis of the right side, Chvostek* merely increased the secretion of perspiration on the right side of the face by galvanising the cervical sympathetic (the cathode being placed in the neighbourhood of the ganglion cervicale supremum, and the anode on the opposite side, near the spinous processes of the cervical and upper dorsal vertebræ). On the other hand, Nitzelnadel,† in a similar case of unilateral ephidrosis, produced a diminution in the secretion by galvanising the sympathetic.

THORACIC AND ABDOMINAL PARTS OF THE SYMPATHETIC SYSTEM.

According to their physiological functions, so far as we know them, we can distinguish the following varieties of branches in the thoracic and abdominal sympathetic nerves:—

1. *Excitomotor branches of the thoracic and abdominal viscera.*
 2. *Movement-controlling (regulating) branches.*
 3. *Vasomotor (or secretory and trophic) branches.*
 4. *Sensory branches, acting reflexively.*
- I. *Excitomotor branches of the thoracic and abdominal viscera.*

1. *The Heart.*—We have already had under consideration the excitomotor nerves of the heart distributed in the cervical sympathetic; and it has also been mentioned that branches pass from the spinal cord to the upper thoracic ganglion and the ganglion stellatum, and thence enter the cardiac plexus. According to V. Bezold and Bever‡ these branches rise partly from loops of the brachial plexus, and partly from the plexus surrounding the vertebral artery; they were not successful in tracing them back to the trunks of the cervical nerves in rabbits. Schmiedeberg, also, could not find excitomotor nerves in the

* "Wiener med. Wochenschrift," 1872, No. 19 and 20.

† "Ueber nervöse Hyperidrosis und Anidrosis," Diss. Jena, 1867.

‡ "Würzburger med. Zeitschrift," 1867, Bd. viii, p. 215; "Untersuchungen aus dem physiologischen Laboratorium in Würzburg," Leipzig, 1867, Heft 2, p. 181.

spinal roots of the first thoracic ganglion in dogs. Besides such branches as are of uncertain origin, the trunk of the sympathetic in its whole length, as far downwards as the lumbar region, contains fibres the irritation of which accelerates and strengthens the contractions of the heart, as Budge and Donders have shown in frogs, and V. Bezold in rabbits. Von Bezold also regarded as excitomotor those filaments which, issuing from the thoracic and lumbar parts of the spinal marrow, pass upwards in the trunk of the sympathetic, and eventually emerge from the uppermost thoracic ganglion as *Nervi cardiaci inferiores*. Nevertheless this, as we now know, is not the correct view; they are rather regulating fibres, conducting impressions centripetally, and acting in a reflex way on the vagus nerve.

2. *Stomach and Intestines*.—Nothing is known with certainty regarding the influence of the sympathetic on the movements of the stomach. The peristaltic movements of the intestines, on the other hand, though automatically excited through the parenchymal ganglia, seem capable of receiving accelerating influences from the thoracic and abdominal parts of the sympathetic system. The inhibitory nerves of peristaltic action, afterwards to be considered, appear to run in the trunk of the splanchnic nerve; at least one circumstance favours that view,—that irritation of the splanchnic in animals arrests the movements of the intestines.

The colon descendens and the rectum receive, according to Nasse,* motor fibres from the plexus surrounding the inferior mesenteric artery.

3. *Urogenital Apparatus*.—The movements of the bladder, the ureter, the seminal vesicles, and the uterus, are partly excited or increased by irritation of the trunk of the sympathetic nerve in the abdomen or of the plexuses in the abdominal cavity; but it is still an open question whether the originating or increasing of these movements takes place directly from the sympathetic by excitomotor fibres, or reflexively through the afferent or sensory nerves of the viscera. As regards the bladder, Budget† has shown that most of its direct motor branches come from the spinal cord, from the centrum genito-spinale superius and inferius, through the sacral nerves; some also pass through the sym-

* "Beiträge zur Physiologie der Darmbewegung," Leipzig, 1866.

† Henle's and Pfeufer's "Zeitschrift für rationelle Medicin," xxi., p. 174.
"Wiener med. Wochenschrift," 1864, No. 39-41.

pathetic hypogastric plexus to the vesical plexus; in the latter, also, are sensory and reflexively-acting fibres.

Regarding the movements of the uterus, opinion varies very much. According to the researches of Obernier,* Frankenhäuser,† and Koerner,‡ the hypogastric plexus and the spermatic nerves arising from it appear to contain all, or the most important, motor nerves of the female genital apparatus; Kehrer,§ on the other hand, got no result whatever from irritation of the plexus hypogastricus (magnus). Kehrer regards the sacral nerves as exclusively motor nerves of the uterus; and Koerner and Obernier at least admit the presence of excitomotor fibres in the sacral nerves; whilst Frankenhäuser believes them to be inhibitory nerves, and considers the inferior mesenteric ganglion the proper motor centre of the uterus. According to the above-named authors, the centre for the uterine spinal nerves is to be sought for partly in the neighbourhood of the last dorsal and four first lumbar vertebræ, and partly further upwards, perhaps even in the brain (the cerebellum?); and these statements are corroborated by the latest experiments of Oser and Schlesinger.

Movements in the vasa deferentia and the vesiculæ seminales followed, according to Budge|| and Loeb,¶ irritation of the trunk of the sympathetic in rabbits, from the ganglion lying on the fifth lumbar vertebra downwards; while irritation above that level was followed by no result. The fibres producing that effect rise, as shown by Budge, in the centrum genito-spinale superius, at the level of the fourth lumbar vertebra.

II. *Movement-Controlling (regulating) Branches.*—Under this title we have to distinguish between two varieties of branches—(a) Those which control the movements in the viscera directly, centrifugally, that is, probably through the operation of automatic exciters of movement—excitomotor ganglia in the parenchyma—*controlling nerves*, in the strictest sense; (b) Those which exercise their function

* "De nervis uteri." Diss. Bonn, 1862.

† "Die Bewegungsnerven der Gebärmutter. *Jena'sche Zeitschrift für Medicin und Naturwissenschaft*, i., 35 and 46.

‡ "De nervis uteri." Diss. Breslau, 1862; "Centralblatt für die Med. Wissenschaft," 1864, No. 23.

§ Ueber Zusammenziehungen des Weiblichen Genitalapparats ("Beiträge zur vergleichenden und experimentellen Geburtskunde." Giessen, 1864).

|| See "Meissner's Jahresbericht," 1858, p. 585.

¶ *Ibidem*, 1865, p. 488.

reflexively, centripetally, by acting on the movement-controlling, regulating mechanisms in the central nervous apparatus—*reflex inhibitory nerves*. The latter should probably also be regarded as sensory nerves; like these, their action is reflex, only that action does not show itself in movement, but rather in checking or controlling movement.

The splanchnic nerve is generally considered a direct inhibitory nerve; Pflüger proved that irritating it arrested the peristaltic movement in the small intestine. Indisputable as this fact is, the inference evidently to be drawn from it—that of a directly-controlling action of the splanchnic—has lately become somewhat doubtful; we know, especially, that the splanchnic is also the vasomotor nerve of the small intestine (see below), and the lessening of the arterial blood supply following the irritation might be regarded as indirectly the cause of the decrease or arrest of the peristaltic action. The controlling fibres of the splanchnic pass, as has been pointed out by Pflüger and Nasse, into the dorsal part of the spinal cord; their central termination, however, has not yet been ascertained.

The reflex inhibitory nerves have an action similar to that of the superior laryngeal on the respiratory movements (F. Rosenthal); those which control the movements of the heart and bloodvessels are contained in the thoracic and abdominal parts of the sympathetic, as Goltz and Bernstein have demonstrated. Goltz* found that, in striking or tapping on the skin of the abdomen in frogs, or on the exposed abdominal viscera, the so-called percussion experiment (Klopfversuch), he produced a temporary diastolic arrest of the heart's action and a hyperæmic dilatation of the vessels of the abdomen. He explained the arrest of the heart's action as a reflex irritation of the vagus—the dilatation of the vessels of the abdomen as paralytic relaxation (atony) due to decreased activity of the vasomotor nerve-centre. Nevertheless he did not succeed in demonstrating particularly the routes by which this reflex action travels. On the other hand, Bernstein† has more lately proved that those branches which stimulate the vagus reflexively run in the trunk of the

* "Archiv. für Pathologische Anatomie," xxviii., p. 428; xxix., p. 294, "Centralblatt für die Med. Wissensch.," 1864, No. 40.

† "Herzstillstand durch Sympathicus-Reizung;" "Centralblatt für die Med. Wissensch.," 1863, No. 52; 1864, No. 16. "Untersuchungen über den Mechanismus des regulatorischen Herznervensystems, Reichert's und du Bois Reymond's Archiv. für Physiologie," 1864, p. 614.

sympathetic, and, for the most part, pass into the spinal cord between the third and sixth dorsal vertebræ through the communicating branches; as we have formerly mentioned, only a few are found further up, as high as the ganglion stellatum. After division of the sympathetic the percussion experiment gives no further result; but the effect of irritation of the sympathetic on the heart remains when both vagi are previously divided, or when the medulla oblongata is destroyed. Further, Bernstein succeeded, at least in frogs, in demonstrating the peripheral branch in which the reflex controlling fibres are conducted from the abdominal viscera to the trunk of the sympathetic. It is a small nerve which accompanies the mesenteric artery; stimulation of this nerve caused the diastolic arrest of the heart's action, as in irritation of the trunk of the sympathetic or of the exposed abdominal viscera. Bernstein states that similar treatment of the splanchnic does not produce the same effect.

Concerning the controlling power of the *nervi erigentes* on the bloodvessels of the penis, see below (vasomotor branches).

III. *Vasomotor, or Secretory and Trophic Branches.*—The vasomotor nerves of the thoracic and abdominal viscera are distributed for the most part, if not entirely, by the sympathetic—the plexuses and peripheral branches of the thoracic and abdominal portions.

1. *The Lungs.*—Formerly the vagus was generally regarded as the vasomotor nerve of the lungs, this view being supported by the well-known changes which take place therein on division of the vagus (Longet, Wundt, Schiff, and others). Traube has shown that the cause of these changes is that portions of food get down the air-passages, and that the vagus, as a vasomotor nerve, has no share in producing them. Wundt* conjectured that the sympathetic branches were concerned in the innervation of the vessels of the lungs; and lately Brown-Séquard† concluded, after experiments on dogs, rabbits, and guinea-pigs, that the vasomotor nerves of the lungs are not distributed in the vagus, but through the spinal cord (cervical portion) and the first thoracic ganglion of the sympathetic.

A carefully-conducted series of experiments performed by

* Versuche über den Einfluss der Durchschneidung der vagi auf die Respirations-organe: Müller's "Archiv für Physiologie," 1855.

† On Ecchymoses and other Effusions of Blood caused by a Nervous Influence: Archives f. Scientific and Practical Medicine, 1873, Febr., No. 2, p. 148.

Bischofswerder* on rabbits led to the conclusion that the vagus, as well as the sympathetic, acts as a vasomotor nerve of the lungs. The simultaneous extirpation of the uppermost thoracic ganglion and both vagi was followed by a much stronger hyperæmia and infiltration of the lungs than division of the vagi only.

According to Brown-Séquard's observations, the vasomotor nerves of the lungs appear to have a cerebral origin. This author repeatedly saw pneumonic infiltration, hæmorrhages, and œdema of the lungs, after injury to the base of the brain. Nothnagel made the same observation after injury to a certain spot on the surface of the rabbit's brain, near the sulcus found on its superior aspect; sometimes nearly the whole lung was infiltrated with blood.

2. *Stomach and Bowel.*—The secretion of the gastric juice seems to be carried on automatically by the ganglia situated in the walls of the stomach. The sympathetic has no direct influence on the process, so far as we know; the vagus, however, does exercise some such influence, but perhaps only reflexively, as the sensory nerve of the stomach. This view is supported by many experiments, amongst others those of Lussana and Inzoni.† The sympathetic plexuses and ganglia have, however, some control over the nutrition of the walls of the stomach; thus, Pincus‡ and Adrian§ observed, after extirpation of the solar plexus, various changes in the mucous membrane of the stomach and upper part of the small intestine (intense hyperæmia, extravasation of blood, and ulceration), but none in the secreting power of the stomach.

The vasomotor nerve of the intestine, at least of the small intestine, and probably of most of the abdominal viscera, is the splanchnic, the principal vasomotor nerve in the body. Irritation and extirpation of the different ganglia and sympathetic plexuses of the abdomen have a certain, but very inconstant influence on the intestinal secretion, the nature of the intestinal evacuations, and on the general nutrition. Thus, after extirpation of the celiac plexus some observers noticed increased secretion from the intestines (Adrian), or

* "Vagus und Sympathicus, die vasomotorischen Nerven der Lunge." Dissertation. Greifswald, 1875.

† "Gaz. Hebomad," 1863, x., 13.

‡ "Exper. de vi nervi vagi et sympathici ad vasa secret. nutrit. tractus intestinalis et renum," Diss., Breslau, 1856.

§ "Ueber die Functionen des plexus coeliacus und mesentericus," Dissert., Giessen., 1861.

hæmorrhagic diarrhœa (Cl. Bernard); Lumansky* also observed emaciation and discharge of undigested food per anum.

The centre for the vasomotor nerves of the intestine, like that of the vasomotor nerves of the liver, &c., appears to be in the brain. We ourselves frequently noticed the occurrence of hæmorrhagic diarrhœa in dogs, after injury to different parts of the cerebellum.

3. *The Liver*.—Schiff first proved that the vasomotor nerves of the liver are distributed in the sympathetic; and he at the same time drew attention to the great importance of these nerves in the pathogeny of diabetes mellitus produced experimentally in animals, by means of Bernard's puncture, &c.

The vasomotor nerves of the liver are held by Schiff † to rise in the neighbourhood of the floor of the fourth ventricle of the brain, the medulla oblongata, and the anterior half of the cervical and thoracic parts of the spinal cord, as far downwards as the fourth or fifth dorsal vertebra in mammalia. Here they enter the sympathetic through the communicating branches, so pass downwards, and eventually accompany the vessels of the liver (as the hepatic plexus) to the interior of the parenchyma of the gland.

He states that injury to these vasomotor nerves in any part of their course has the same effect as wounding their centre by puncture. In all these cases the first result was a paralytic dilatation of the vessels of the liver producing considerable hyperæmia. It is this latter which, by transformation of the glycogen, causes the formation of sugar, which appears in the blood and afterwards in the urine.

Pavy ‡ has noted the occurrence of diabetes after injuring the uppermost cervical ganglion; Eckhard, § after destroying the uppermost thoracic and inferior cervical ganglia, after wounding the vermiform process of the cerebellum in rabbits, and after section of the spinal cord from the medulla oblongata downwards to the level of the lumbar vertebræ; and, finally, Schiff observed it after division of the sciatic nerves. These experiments, and the occurrence of a transient melituria after each considerable disturbance

* "Zeitschrift für rationelle Medecin," Band xxviii., p. 259 (1866).

† "Journal de l'Anat. et de la Phys.," 1866, p. 354.

‡ "Researches on the Nature and Treatment of Diabetes." London, 1862.

§ "Beiträge zur Anatomie und Physiologie," Band iv., p. 3; Band vi., Heft 2.

of the circulation, after ligature of the larger vessels, and under the influence of certain poisonous substances, &c., are probably to be explained on the ground that, under the circumstances mentioned, either by direct or indirect co-operation of the vasomotor nerves of the liver, there is an increased flow of blood to that organ, and, consequently, an increased production of sugar. This explanation is further supported by the later experiments of Cyon and Aladoff,* who also bring to light many new details on this subject. They established the fact that extirpation of the cervical ganglia of the sympathetic, and of the lower ganglion alone, immediately and constantly produces diabetes. The same result follows even when the ganglion is carefully, without any direct disturbance, raised and allowed to remain in its place, and then all the nerve-branches, both central and peripheral, with which it is connected, divided. It follows from that that the diabetes is not produced, as Eckhard believed, by stimulation, but by a paralysis of the sympathetic nerves; and that the nerves, paralysis of which causes diabetes, stand in connection with the last thoracic and first cervical ganglia. An examination of the afferent and efferent nerves of these ganglia, with respect to their influence on the formation of sugar, showed that diabetes occurs when division is practised either on both rami vertebrales or on both the nerves which pass from the inferior cervical ganglion to the first thoracic ganglion, encircling the subclavian artery and forming the so-called annulus Vieussenii. On the other hand, division of the other nerves of the inferior cervical ganglion causes no diabetes. Thus the nerve-fibres, paralysis of which produces diabetes, leave the spinal cord through the rami vertebrales, traverse the ganglion cervicale inferius, and pass thence in the trunk of the sympathetic and in the splanchnic nerves to the liver. One might thus expect that division of the splanchnic nerves should occasion diabetes. In fact, V. Gräfe,† Hensen, and Ploch,‡ had observed, amongst other things, the occurrence of the disease after division of the splanchnic; Eckhard§ and Pavy deny this, however, and even state that, after preliminary division

* "Bulletin de l'Acad. Imperiale des Sciences de St. Petersbourg," 1871. Tome xvi., p. 308.

† See "Krause, Annotationes ad Diabetem." Halle, 1858.

‡ "Ueber den Diabetes nach Durchschneidung des N. Splanchnicus," Dissert., Giessen., 1863.

§ "Beiträge zur Anatomie und Physiologie," 1867, Band iv., p. 3.

of the splanchnic, "Bernard's puncture" does not cause diabetes. The further statements of Cyon and Aladoff are corroborative of these. They found that division of the trunk of the sympathetic between the tenth and twelfth ribs in dogs almost never produces diabetes; and further; that when this section has been performed, division of the lowest cervical and uppermost thoracic ganglia is no longer followed by diabetes. When, on the other hand, the splanchnic or the trunk of the sympathetic is divided only after the "puncture" (pricking the floor of the fourth ventricle with a needle), or after wounding the ganglia just mentioned, the artificial diabetes remains.

A solution of these apparent contradictions is possible only on the ground that certain nerves, paralysis of which hinders the occurrence of diabetes in some way, enter the trunk of the sympathetic below the first thoracic ganglion. Thus there must be two sorts of fibres in the lower part of the sympathetic and in the splanchnic, of which the one variety (those coming from the first thoracic ganglion, hepatic vasomotor nerves), when paralysed, produces diabetes; while the other, emerging from the spinal cord at a lower level, prevents diabetes when paralysed. These latter fibres are merely, as Cyon and Aladoff have shown to be very probable, the vasomotor fibres of other organs. In order to the production of diabetes there must be a greater flow of blood through the dilated hepatic vessels, as, in fact, occurs in paralysis of the vasomotor nerves of the liver, contained in the annulus Vieussenii. After division of the lower part of the sympathetic, or of the splanchnic, there is an accumulation of blood in the other organs, whereby the augmentation of the quantity of blood in the liver in consequence of the simultaneous paralysis of *its* vasomotor nerves is reduced to a minimum. If, on the other hand, by preliminary "puncture," or by extirpation of the uppermost thoracic ganglion, dilatation of the hepatic vessels has already occurred, division of the splanchnic cannot, at least at first, put an end to this dilatation; thus the diabetes, after this operation, lasts some time.

Budge* observed increase of size and congestion of the liver also after extirpation of the semilunar plexus. This was not, however, confirmed by Adrian.† Moreover, this

* "Verhandlungen der Leopold. Carol. Academie," Band xix., p. 258.

† Eckhard's "Beiträge zur Anatomie und Physiologie," 1862, Bd. iii, p. 61.

proceeding often leads to the early death of the animal (within 24 hours), through injury to the neighbouring parts.

4. *The Kidneys.*—These organs obtain their vasomotor nerve supply partly from the sympathetic and partly from the splanchnic nerves. This is proved by certain experiments in which various qualitative and quantitative changes in the urine (apart from Glycosuria), and disturbances in the nutrition of the kidneys and suprarenal capsules, take place after interference with the sympathetic.

It is a noteworthy fact that the diabetes occurring after dividing the sympathetic is usually conjoined with hydruria; but diabetes without hydruria, or hydruria without diabetes, may be set up according as the vasomotor nerves of the liver and kidneys are interfered with in the joint or separate part of their course. Thus Cyon and Aladoff, in their fore-mentioned experiments, observed only diabetes without hydruria. Knoll,* after division of the splanchnic, only polyuria without diabetes. The experiments were performed in such a way that cannulæ were placed in both ureters in dogs, the splanchnic on one side divided, and the quantities of urine secreted on each side compared. On the side operated on the quantity was considerably increased. Eckhard, after mechanical irritation of the vermiform lobe of the cerebellum in rabbits, noticed melituria; but after previously dividing the hepatic nerves, only hydruria. Bernard and Donders discovered that, on injuring a certain spot in the fourth ventricle of the brain, the secretion of urine was increased; this spot is very near to, but does not coincide with, that part injury to which produces diabetes. On the whole, these experiments support the view that the vasomotor, or secretory, nerves of the kidneys, like those of the liver, have a cerebral centre. Other changes in the urine (albuminuria, hæmaturia), indicating more serious alteration in the action of the kidneys, have been met with by other investigators, after experiments in which not only the renal nerves in their course were operated on, but also certain parts at the base of the brain. Bernard holds, with regard to secretion in the kidneys, that there is a nervous antagonism between the vagus and splanchnic similar to that which exists in the large salivary glands of the mouth between the nerves of secretion distributed in the sympathetic and those of the chorda tympani. Thus, on irritating the vagus there is said

* "Ueber die Beschaffenheit des Harns nach der Splanchnicus-durchschneidung" (Eckhard's "Beiträge zur Anatomie und Physiologie," 1871).

to occur an increased flow of blood, distension of the veins, a lighter colouration of the venous blood, and increased secretion: irritation of the splanchnicus major, on the contrary, is said to produce decrease of the flow of blood and of the secretion, and a darker colouration in the venous blood.

That also the vasomotor nerves of other abdominal organs (as the spleen) and of the whole genital apparatus are included in the trunk of the sympathetic is undeniable from the anatomical point of view, although special investigation by experiment has hitherto given but vague results. Irritation of the splenic plexus, and of a branch of the cæliac plexus, according to Jaschkowitz,* reduces the size of the spleen, probably from contraction of the vessels in this very vascular organ. On the other hand, it has been proved by Bochefontaine† and von Tarchanoff‡ that after ligature of the splenic plexus, or division of the splenic nerves, a distension of the spleen occurs, which is brought about by a dilatation of the vessels; in a few days this subsides, exactly as in the case of the vessels of the ear after division of the cervical sympathetic. As for the vessels of the penis, we know, from the researches of Löven, that irritation of the nervi evigentes results in relaxation of the arteries. It is still, however, undecided whether this is really produced by vessel-dilating nerves or by a reflex lowering of the arterial tone by the irritation of sensory nerves, as in Goltz's percussion experiment.

In the sympathetic are distributed, not only the vasomotor nerves of the thoracic and abdominal viscera, but also *those of the extremities*. Claude Bernard§ believed that the vasomotor fibres for the upper and lower limbs do not at first form part of the plexuses of the extremities, but, coming from the trunk of the sympathetic, join them through the rami communicantes. The investigations of Schiff|| also tend to indicate that the vasomotor nerves of the upper extremities come partly from the spinal cord through the communicating

* "Dissertation," Berlin, 1857.

Brown-Séquard's "Archiv," t. vi, 1874, p. 698.

† Pflüger's "Archiv für Physiologie," Band viii., p. 97, 1874.

§ "Comptes rendus," 1862, II, p. 228, 400, 425; "Journ. de la Phys.," V. 33.

|| "Neurologische Untersuchungen," Frankfort a/m., 1855:

branches and join the thoracic part of the sympathetic, while those for the lower extremities proceed to the sacral portion; and, further, that there are also vasomotor nerves of spinal origin which do not traverse the sympathetic, but pass onwards directly in the spinal plexuses and nerve-trunks. The brachial plexus thus conveys the vasomotor nerves destined for the skin and muscles of the upper limbs (Schiff); the skin of the trunk is supplied from the dorsal and lumbar nerves, that of the lower extremities from the lumbar and sacral plexuses (Pflüger, Schiff). It is only for the lower part of both limbs that vasomotor fibres pass directly from the trunk of the sympathetic, to which they were conducted through the anterior roots of the spinal nerves.

IV. *Sensory branches (acting reflexively).*—Whilst the sensory fibres for the thoracic viscera (the heart and lungs) are almost exclusively connected with the vagus, those for the abdominal viscera are, for the most part, distributed by the sympathetic system. It is for the stomach only that the vagus is to be regarded with certainty as a nerve of sensation, as the forementioned experiments of Lussana and Inzoni demonstrate. It has not yet been shown whether, in addition to it, sympathetic fibres (branches from the solar plexus) take part in the sensory innervation of the stomach, as has been often asserted on pathological grounds.

The sensory nerves of the *Intestine* appear to proceed from the splanchnic nerves. Ludwig and Haffter* found that division of these nerves produced very great pain, an observation which Nasse† corroborates. Whilst the motor and inhibitory fibres of the splanchnic supply only the small intestine, the sensory fibres, according to Nasse, reach the ascending and transverse colon; the descending colon and the rectum receive motor and sensory fibres from the plexus which surrounds the inferior mesenteric artery. The latter may also include sensory filaments for the walls of the bloodvessels; Colin,‡ at least, holds that the arteries of the abdominal viscera are characterised by great sensibility, which the arteries of the superficial parts do not possess.

In passing we shall here mention that formerly, when a greater degree of independent action was generally attri-

* "Neue Versuche über den N. Splanchnicus major and minor, diss. Zurich," 1853. "Henle's und Pfeufer's Zeitschrift für rationelle medicin. N. F." Band iv. p. 322.

† "Beiträge zur Physiologie der Darmbewegung." Leipzig, 1866.

‡ "Sur la Sensibilité des artères viscérales, comptes rendus," lv., p. 403.

buted to the sympathetic nervous system, even by eminent physiologists (Bidder,* Volkmann†), it was regarded as the centre not merely of motion, but also of sensation, for all the vegetative organs of the body. It is clear that at the present day we can no longer accept this view; and just as little importance can be attached to that of Küttner and Volkmann, regarding the possibility of a "Cross-conduction" ("Quer-leitung,") by transference of sensory impressions from the sympathetic to cerebro-spinal fibres. We can now only regard it as possible that sensory filaments pass through the sympathetic to the cerebro-spinal system, notwithstanding that these cerebro-spinal offshoots, and the central terminations of the sensory fibres of the sympathetic (as, for instance, those contained in the splanchnic), are still, to a great extent, unknown to us.

Budge‡ has given us some reliable data regarding the sensory nerves of the *bladder*. According to him the sensory or reflex nerves of the bladder and urethra emerge from the spinal cord with the 3rd and 4th sacral nerves, and join the hypogastric plexus and lumbar part of the sympathetic. Irritation of the latter produces reflex contractions of the bladder, which do not appear after the third and fourth sacral nerves have been divided. Gianuzzi§ also, by irritating the branches passing from the sympathetic to the hypogastric plexus, obtained contractions of the bladder, occurring more slowly, and only after more powerful irritation, than when the second, third, and fourth sacral nerves were operated on.

Besides the fibres which are, properly speaking, sensory (amongst which we, according to the present state of opinion, can only reckon such as pass upwards to the brain, and make their condition of irritation known by distinct sensation), there is still a large number of centripetal, reflexively-acting filaments in the trunk of the thoracic and abdominal sympa-

* "Erfahrungen über die functionelle Selbständigkeit des sympathischen Nervensystems. Müller's Archiv. für Physiologie," 1844, p. 359.

† "Die Selbständigkeit des sympathischen Nervensystems durch Anatomische Untersuchungen nachgewiesen," Leipzig, 1842. Kölliker assumed an intermediate position: "Die Selbständigkeit und Abhängigkeit des sympathischen Nervensystems durch anatomische Beobachtungen bewiesen," 1845.

‡ Henle's und Pfeufer's "Zeitschrift für rationelle Medicin." Band xxi. und xxiii; "Wiener med. Wochenschrift," 1864. No. 39-41.

§ See Meissner's "Jahresbericht," 1863, p. 404.

thetic. We have already discussed those fibres which have a reflex influence on the heart's contraction, the vascular tone, and the movements of the urogenital apparatus, and have also stated that, as regards many apparently automatic movements in the viscera, it is doubtful whether they are not rather produced, or increased, by the reflex action of centripetal nerves. This refers, for instance, to the peristaltic action of the intestine, the reflex origin of which was asserted by Henle*, in 1840; the seat of this action he, after a series of experiments on mammals, believed to be the ganglia scattered among the intestinal nerves, a statement corroborated by various other experimenters (Budge,† Koelliker‡). Volkmann,§ Longet,|| and Pickford,¶ however, disputed the validity of these conclusions, at least in so far that they found the existence of the spinal cord in its whole length necessary to the production of the more extensive movements in the intestines. The later and frequently-quoted experiments of Nasse are partially favourable to the possibility of a reflex origin through interposed intestinal ganglia.

Brown-Séquard and Remak** discovered that division of the sympathetic below the uppermost lumbar ganglion, or of the celiac plexus in frogs and mammals, at once produced persistent dilatation of the pupil on the side operated on. This phenomenon must obviously be understood to be of reflex origin, though the path by which this reflex nervous action travels is still quite unknown.

* "Pathologische Untersuchungen," 1840, p. 92; "Allgemeine Anatomie," p. 724.

† "Untersuchungen über das Nervensystem." Heft. Frankfurt, 1842, p. 178.

‡ "Die Selbständigkeit und Abhängigkeit des sympathischen Nervensystems," p. 34.

§ Müller's Archiv," 1838, p. 29.

|| "Anat. et Phys. du système nerveux," ii., p. 577.

¶ "Archiv. für phys. Heilkunde," 1843, p. 422.

** "Neue Beiträge zur Lehre von der Tabes, Berliner klin. Wochenschrift," 1864, No. 41.

PART II. PATHOLOGY.

In this second part of our Essay we shall discuss a number of diseases which are certainly or probably connected with the sympathetic nervous system.

This connection may result—

1st. From the presence of symptoms which resemble, or are identical with, those which we recognise as expressions of the physiological function of the sympathetic, and which we have enumerated and explained in the former part of this essay.

2ndly. From pathological anatomical changes in the sympathetic nerve.

Those conditions usually go together, but not invariably; thus, the pathological alterations may be completely wanting, or, on the other hand, they may be found in cases which were not before suspected to have any connection with the sympathetic system.

We begin with the consideration of the affections of the cervical sympathetic, afterwards taking up those which probably have relation to some change in the thoracic and abdominal portions of that nerve.

I.—FUNCTIONAL DISTURBANCES IN THE DOMAIN OF THE CERVICAL SYMPATHETIC.

These belong to the most sharply defined group of phenomena met with in the otherwise somewhat obscure pathology of the sympathetic nervous system. The appearances are sometimes those of irritation, sometimes those of paralysis—approaching in character the symptoms either of experimental stimulation or of division of the cervical sympathetic, the latter* being more common than the former. The symptoms of paralysis of the sympathetic in men are rarely

* In Poiteau's comparative observations (*"Archiv. gen. de Med."* 1869, Août) there are nineteen cases of paralysis and nine of irritation of the cervical sympathetic.

so pronounced as those artificially produced in animals by division, as paralysis seldom attacks *all* the fibres of the nerve, vasomotor as well as oculo-pupillary, as is the case in division.

The conditions which give rise to disturbances of function in the cervical sympathetic may, so far as they are known to us, be arranged as follows:—

- (a) Compression of the cervical sympathetic by tumours.
- (b) Injuries to the cervical sympathetic.
- (c) Injury or disease of the cervical part of the spinal cord.

Besides, there occur simple functional disturbances in the cervical sympathetic, either alone or accompanying some other disease, and without any objectively-demonstrable cause.

In the above order we will now shortly submit the following observations:—

A. *Compression of the Cervical Sympathetic Nerve by Tumours.*—Willebrandt* observed, in cases of glandular swelling in the neck, a contraction of the pupil, which returned to its normal size when inunction of iodide of potassium had caused the tumours to subside. In cases reported by Ogle,† Heineke,‡ and Verneuil,§ contraction of the pupil was brought on by large carcinomatous growths in the neck. In the last quoted case there were also symptoms showing that the vasomotor filaments were involved—elevation of temperature and increased secretion of perspiration on the whole of one side of the face. In another case recorded by Ogle,|| the vasomotor and oculo-pupillary symptoms were very well marked, the compression of the sympathetic being caused by a cicatrix on the right side of the neck. There were here, on the right side, contraction of the pupil, flattening of the cornea, injection of the conjunctiva, congestion of the ear and cheek, dilatation of the temporal artery, and elevation of temperature in the cavities of the

* Willebrandt, "Archiv. für Ophthalmologie," 1855, Bd. I., p. 319.

† Ogle, "Medico-Chirurgical Transactions," T. xli., p. 398.

‡ Heineke, "Greifswalder med. Beiträge," 1860, Bd. ii.

§ Verneuil, "Gaz. des hôpitaux," 1864, 16 April.

|| Ogle, "Lancet," 17 April, 1869.

mouth and nose. The same phenomena occurred in a case reported by du Moulin.* B. Fränkel† has lately put on record a case of compression of both sympathetic nerves and of both vagi by a large glandular swelling in the neck, in which the vasomotor and oculo-pupillary fibres were incompletely paralysed. Compression of the sympathetic is also sometimes caused by aneurism of the aorta, of the innominate arteries, and of the carotid: such cases have been observed by Gairdner, Coates, and others, and in one of them the contraction of the pupil disappeared after ligature of the carotid.

Those causes which bring about a state of paralysis in the sympathetic may also produce a condition of irritation, together with all the phenomena dependent on such a condition; and according as only the oculo-pupillary, or also the vasomotor fibres, are irritated, we have dilatation of the pupil alone, or also pallor and a decrease of temperature on the corresponding side. Ogle‡ has published several such cases, especially one communicated to him by Kidd, which is interesting on account of the varying character of the phenomena—at one time those of irritation, at another those of paralysis. It was a case of acute abscess in the neck in which, simultaneously with the formation of pus, and accompanied by acute pain and shiverings, there was an extreme degree of *dilatation of the pupil*, which disappeared after the patient had had a quiet sleep. On the following day the shiverings were renewed, and accompanied by *contraction* of the pupil: this was succeeded by a paroxysm of pain, in which, as at first, the pupil became *dilated*. The same variations were repeatedly observed; and when the abscess had been opened, and was progressing towards cure, the pupil assumed its normal dimensions. In the following year the lady had another abscess in the same region, and in the year after that a third, more deeply situated in the tissues, and all accompanied, but to a less degree, by the same train of symptoms. The phenomena in this apparently complicated case are easily explained. The compression, occasioned by the inflammatory exudation and suppuration, acted first as a stimulant to the pupillary fibres of the sympathetic, hence the mydriasis; on returning, or on being

* Du Moulin, "Bullet. de la Soc. de Méd. de Gand," 1872.

† Fränkel, "Berliner klinische Wochenschrift," 1875, No. 3.

‡ Ogle, "Med. Chir. Transactions," T. xli., p. 398.

increased in intensity, this compression had the effect of a force which lessens conducting power—thus causing myosis. Possibly also the rigors may be traced to an occasional abnormal excitation of the vasomotor filaments in the cervical sympathetic. When the bloodvessels of one side of the head contract, less blood flows to the medulla oblongata; this temporary anæmia stimulates its vasomotor nerve-centres, by which means the phenomena attending rigors (contraction of the smallest arteries of the skin) are developed.

H. Demme* noticed mydriasis and slight exophthalmos in a man suffering from cystic goître. At the examination *the cervical part of the sympathetic on the left side was found abnormally red*, and surrounded by a serous exudation in the connective tissue. No changes were found on microscopic examination. Clearly the swelling here had irritated the oculo-pupillary fibres of the sympathetic. We have met with a very similar case—that of a patient suffering from a vascular goître almost entirely confined to the right side. The symptoms were extreme mydriasis, complete immobility of the iris, considerable exophthalmos, and loss of accommodation power, in the right eye; in addition to these there was a persistent lowering of the temperature of the auditory meatus of the same side, amounting to $0.3-0.4^{\circ}$ C. compared with the healthy side. The pulse was much accelerated. Calabar bean effected a temporary improvement in the mydriasis and the accommodative paralysis; local galvanopuncture, also, was followed by a slight decrease in the size of the tumour and in the frequency of the pulse.

We have thus cited instances, first of paralysis, then of irritation, of the sympathetic, brought on by the pressure of tumours. In both groups the phenomena in the domain of the oculo-pupillary fibres (myosis, connected with paralysis, and mydriasis with irritation) are of a constant and persistent nature, while in the domain of the vasomotor fibres (reddening or pallor) they are inconstant and transient; the reasons for this are yet unknown. Vulpian† supposes that there are vasomotor symptoms in every case of affection of the sympathetic, but only at the beginning; that they sometimes disappear in a few days, or even hours. If such patients come under medical observation only at a later

* Demme, "Würzburger med. Zeitschrift," 1862, Bd. iii., p. 262 and 269.

† Vulpian, "Leçons sur l'appareil vasomoteur." Paris, 1875, p. 142.

period, the vasomotor symptoms may no longer be recognizable, and this, thinks Vulpian, has led to the erroneous conclusion that these symptoms are frequently entirely wanting. There are many considerations in favour of such a view; we would also add that the vasomotor symptoms, when they have disappeared, may periodically reappear.

B. *Injuries to the Cervical Sympathetic*.—The number of exact observations bearing on this point is very small; there are, especially, almost no recorded cases in which we can assume with confidence the existence of a direct, uncomplicated injury, confined to the cervical part of the trunk of the sympathetic.

We will first allude to the following case, which has been published by three American practitioners (Weir Mitchell, Morehouse, and Keen) in their interesting monograph on injuries of nerves.* It was a case of *gunshot wound of the right sympathetic*. The ball had entered on the right side of the neck, 4 centimeters behind the ramus of the jaw, at the anterior margin of the sterno-mastoid muscle; it had passed through the neck, and emerged immediately below, and about $1\frac{1}{2}$ centimeter in front of the angle of the jaw on the left side. The wounds were healed in six weeks, but it was only at the end of ten weeks that the patient came under the observation of these writers, though a comrade of the wounded man had already noticed the *unusually small size of the right pupil* one month after the injury. Besides this, when the patient was first seen, there were observed in the *right eye myopia, slight ptosis, apparent sinking of the outer angle, decrease in the apparent size of the eyeball, and redness of the conjunctiva*. Further, it was repeatedly noticed that, after violent exercise, *the right half of the face was unusually red*, while the left side remained pale; this was accompanied by pain and reddish flashes of light in the right eye. When the temperature was taken after the patient had rested, there was found to be no difference in the mouth and ear on both sides; no observation was made when the patient was excited by exertion. He was able to return to service five months after receiving the injury.

This case, while it probably indicates bruising (or laceration?) of the right sympathetic by the projectile, gives rise to several remarks regarding the symptoms. We find in it

* S. Weir Mitchell, George R. Morehouse, and William Keen—"Gunshot Wounds and other Injuries of Nerves." Philadelphia, 1864.

nothing which does not receive a sufficient explanation in the results of experimentally injuring the sympathetic in animals and from other pathological observations in men. The ptosis, the sinking of the outer angle of the eye, the apparent small size of the right eyeball, all arise from loss of power in certain of the muscles of the eye, which have their nerve-supply from the cervical sympathetic, and whose special functions we have already discussed. The reddening of the conjunctiva and the flow of tears depend on functional derangement of vasomotor nerve fibres which pass from the sympathetic to the first of the branches of the trigeminus, and which supply the conjunctival vessels: paralysis of these is followed by partial relaxation of the vessels, and increase in the flow of blood and of the conjunctival mucous secretion. The altered refractive power of the eyeball, the myopia, is, in the first place, the necessary consequence of the persistent paralytic myosis, and, secondly, may be regarded as produced by the presumed direct influence of the sympathetic on the muscle of accommodation (M. tensor choroidea), or on detached fibres of that muscle.

Alongside of this case may be put one of Kämpf's.* He brought before the Society of Physicians in Vienna a soldier with myosis paralytica in the right eye, following *a wound of the cervical part of the sympathetic of the same side*. This soldier, in the battle of Orleans, received a stab in the right side of the neck, the direction of which was clearly marked by a cicatrix lying on the outer edge of the sterno-mastoid muscle. The myosis, evidently due to paralysis of the fibres of the sympathetic and to the preponderating influence of the sphincter iridis, was in no way improved by the repeated employment of electricity. Kämpf's communication contains no further reference to anomalies in the domain of the cervical sympathetic.

Here, also, should be mentioned a case recorded by Seeligmüller,† in which, as the result of a *gunshot wound*, there was *paralysis of the left cervical sympathetic*, and of the ulnar nerve. During the Franco-Prussian war an officer, twenty-five years of age, was wounded in the left shoulder by a chassepot bullet. The orifice of entrance was on the clavicular portion of the left sterno-mastoid muscle, 3 centimeters above the upper edge of the collar bone; the orifice

* Kämpf, Sitzung der K. K. Gesellschaft der Aerzte in Wienam, 8 März. 1872.

† Seeligmüller, "Berliner klin. Wochenschrift," 1872, No 4.

of exit was to the left of and close to the spinous process of the fourth dorsal vertebra: the first was cicatrized in six weeks, and the latter in eleven. Nine months after receiving the wound, the following appearances were noted: the left palpebral fissure was smaller than the right; the left pupil more contracted than the right, and dilating to a less degree when shaded; injection of the vessels of the conjunctiva and redness of the cheeks, usually equal on both sides, but sometimes more marked on the left side after mental disturbance; the temperature in the left auditory meatus 0.1° C. higher than in the right; a flow of tears from the eyes; *striking emaciation of the left cheek, which appeared flatter than the right*; besides all this a paralysis of the ulnar nerve.

The flattening and emaciation of one side of the face observed in this case, which also occurred in some others to be mentioned further on, appear to occur more frequently at a later stage in paralysis of the sympathetic. At least, Nicati* states, as the result of some original investigations, that to the first stage of the paralysis a second succeeds, characterised by atrophy, pallor, lowering of temperature, and arrest of the transpiration, on one side of the face. We will again refer to this atrophy when discussing unilateral, progressive, facial atrophy, and its relations to lesions of the cervical sympathetic.

Continuing the argument regarding injuries of the sympathetic, we quote a case described by Bernhardt†. On the 4th day of August, 1870, in the war with France, a German soldier was wounded in the left side of the neck by a bullet. Two years after the injury there was rather a large cicatrix, painful on pressure, situated on the front of the neck, at the inner edge of the left sterno-mastoid muscle, and about two fingers'-breadth above the left sterno-clavicular articulation; this was the orifice of entrance of the bullet. The orifice of exit was behind, at the level of the fourth dorsal vertebra, and to the left of the spinous process; here, also, the scar was very tender.

Besides various phenomena depending on a simultaneous injury sustained by the spinal cord, and of which we need give no particular account here, there were the characteristic signs of *paralysis* of the sympathetic, clearly caused by *injury*. The left eye appeared smaller, and less widely open;

* Nicati, "La Paralysie du nerf sympathique cervical." Lausanne, 1873.

† Bernhardt, "Berliner klin. Wochenschrift," 1872, No. 47 and 48.

the left pupil more contracted, and acting more sluggishly, than the right. The left side of the face was more emaciated, at times redder, and always warmer, than the right. Lachrymation was more easily excited in the left eye, and the temperature in the left auditory meatus was $1\frac{1}{2}^{\circ}$ C. higher than in the right.

X Occasionally, though rarely, one meets with the same symptoms resulting from injury that are observed to follow experimental *irritation* of the cervical sympathetic. Seeligmüller* has described such a case. A smith sustained a very severe blow with an iron implement in the left supraclavicular region, so that he lay stunned several hours. Two days later, on coming under medical treatment, it was seen that the left pupil was much dilated, at least one half wider than the right, but still responding quite as readily to the stimulation of light. The left palpebral fissure was somewhat wider than the right, and the left eyeball pushed a little forward. The whole left side of the neck and head (the latter being bald) and the left ear were markedly paler than the corresponding regions on the right side. Pulsation in the right temporal artery was well marked, that on the left scarcely perceptible. The temperature in the left auditory meatus was lower than in the right, the difference sometimes amounting to $0.15-0.9^{\circ}$ C. In the left supraclavicular region was a doughy swelling, which disappeared in a short time. The cervical part of the sympathetic at the inner edge of the sterno-mastoid muscle was, from its middle to the ganglion supremum, very tender. On exercising pressure on the ganglion, and on galvanic irritation of the left sympathetic, the pupil became still more dilated, while pressure on the right sympathetic gave no result. Refraction and accommodation in the left eye remained normal, which proves that the dilatation of the pupil was caused by irritation of the sympathetic, and not by paralysis of the oculomotorius. In about five weeks all the symptoms had passed off, the vasomotor phenomena having lasted only a few days.

A second case, having a bearing on this part of our subject, was reported, in Flensburg, in 1864. In the war with Denmark at that time a soldier was wounded in the neck by a rifle bullet, and immediately afterwards there was decided dilatation of one pupil. Whether, in this case (of which we

* Seeligmüller, "Archiv. für Psychiatrie und Nervenkrankheiten," 1875, Bd. v., p. 835.

have received but a very imperfect report), there were other appearances indicating a state of irritation of the sympathetic, we do not know. The patient died of exhaustion, after prolonged suppuration; at the post-mortem examination it was impossible to find the sympathetic on account of the great destruction of soft parts in the neck.

C. Injury to the Spinal Cord in the Neck and to the Brachial Plexus.—Disturbances of function in the domain of the cervical sympathetic, associated with injuries of the cervical part of the spinal cord possess no special or peculiar character, as both the oculo-pupillary and vasomotor nerve filaments run some distance in that part of the spinal cord, leaving it through the anterior roots and the rami communicantes, and so passing over to the trunk of the cervical sympathetic. As in cases in which the cervical sympathetic itself is wounded, so after injury of the spinal cord in the neck myosis paralytica is often met with, from an interruption (of traumatic origin) of the conducting power of the oculo-pupillary fibres in the medullary part of their course: more rarely this is followed by irritative dilatation of the pupil—mydriasis spastica. The number of observations relating to this point is indeed few, as, in most recorded cases of injury of the spinal cord in the neck, the pupillary symptoms and the local disturbances of the circulation have received no special attention. In about one hundred cases of such injury Rendu* found but sixteen in which notice was taken of the state of the pupil. He has published two of Desormeaux' cases in which, coincident with injury of the spinal cord in the neck, there were various changes in the pupil. In the first case the spinal cord was completely disorganised by dislocation of the sixth cervical vertebra: during life both pupils were greatly contracted, the face and neck of a purplish red hue, the ears very red, while there was no change in the colour of the rest of the body. Thus, in this case, the vasomotor fibres for the vessels of the head were also probably paralysed. In the second case there was hæmorrhagic softening of the spinal cord at the level of the seventh cervical vertebra, produced by fracture in that situation; the patient showed great pallor, *one pupil widely dilated*, and the other somewhat contracted. Here the irritative phenomena seem to

* Rendu, des troubles fonctionnels du grand sympathique, observés dans les plaies de la Moëlle cervicale. "Archiv. gen. de méd.," 1869, Sept., p. 286-297.

have predominated, as may be inferred from the facial pallor and the mydriasis.

In one case of injury of the spinal cord, by a fracture of the seventh cervical vertebra, Hutchinson* observed contraction of the pupil—a symptom which was wanting in two otherwise similar cases.—Finally, we quote a case of injury of the spinal cord, recorded by M. Rosenthal,† which occurred in the person of a tradesman who had been stabbed in the neck, in the neighbourhood of the sixth cervical vertebra. Besides paralysis of the upper and lower limbs of the right side, he found *dilatation of both pupils*, especially of the left, and a *strikingly slow pulse* (48 per minute), which phenomena lasted four weeks. The patient eventually completely recovered. Here, as in the second of Rendu's cases, mentioned above, there existed a condition of irritation in the pupillary filaments of the sympathetic, accompanied also by a similar affection of the cardiac fibres of the vagus.

Paralytic myosis occurs not uncommonly, also, in non-traumatic diseases of the cervical portion of the spinal marrow, when the oculo-pupillary fibres are affected. Thus, Ogle has observed contraction of the pupil in five cases of disease of the spinal cord in the neck; and lately Bäerwinkel‡ has described paralysis of the oculo-pupillary sympathetic fibres as a symptom in sclerosis of the medulla oblongata. In certain forms of tabes dorsalis, especially in that called tabes cervicalis by Remak, contraction of the pupil, on one or both sides, is a very characteristic symptom. It has been shown that paralysis of the oculo-pupillary filaments takes place in some cases of progressive muscular atrophy: to this we will again refer. On the other hand, it happens less frequently that irritative phenomena (such as dilatation of the pupil) result from non-traumatic affections of the spinal marrow in the neck. We ourselves have met with a case of that nature—mydriasis spastica from caries of the vertebræ. The patient was a boy of eight years, suffering from Potts' disease of the last cervical and three upper dorsal vertebræ. The right pupil was always wider than the left, and responded but very feebly to the stimulus of light, while the left still preserved its normal sensibility. The diameter of the right pupil was $3\frac{1}{2}$ lines, that of the left only 2 lines. The degree of mydriasis present was not the greatest pos-

* Hutchinson, "Lancet," 1875, 21 and 29 May.

† M. Rosenthal, "Oesterr. Zeitschrift für pract. Heilkunde," 1866, No. 46.

‡ Bäerwinkel, "Archiv. für klin. medicin," Band xiv., p. 545.

sible, as instillation of atropine dilated the left pupil to the extent of more than 4 lines; the immobility of the pupil, also, was not complete, as a very powerful light still produced a slight contraction. A low degree of hypermetropia was found on both sides, especially the right; accommodation and definition were normal. No constant or important differences in the action of the vasomotor nerves of the two sides of the head could be recognised. On ophthalmoscopic examination it was found that in the fundus of the eye, otherwise normal, there was a complete equality in the size of the arteries and veins. The spastic mydriasis remained about four weeks, and then gradually disappeared. When the patient was dismissed, after three months' treatment, both pupils were again nearly equal, the right, however, acting more sluggishly than the left.

The cause of the mydriasis in this case can be sought only in a morbid change in the spinal cord and its immediate surroundings, and consequent irritation of the pupillary sympathetic fibres; it is to be looked for particularly in the inflammatory processes in the bodies of the vertebræ, involving just those vertebræ (the lower cervical and upper dorsal) which correspond to the centrum ciliospinale inferius. Whether it is from simple compression, or from inflammation going on in the cord and its membranes, with thickening of the latter, and with circumscribed softening, are questions which must remain undecided.

Hutchinson* first observed also that in cases of *traumatic paralysis of the brachial plexus* there is usually a simultaneous *paralysis of the cervical sympathetic*, manifesting itself by myosis with loss of mobility of the pupil, by narrowing of the palpebral fissure, and by a rise in the temperature of the corresponding side of the face. Seeligmüller† afterwards confirmed Hutchinson's statements. Two instructive cases of traumatic paralysis of the brachial plexus with functional disturbances, especially in the domain of the oculo-pupillary fibres, of the cervical sympathetic came under his observation. In the first case, that of a child aged nine months, who had sustained a fracture of the clavicle and of the neck of the scapula in the act of birth, there was complete paralysis of motion and sensation in the right forearm; there were, moreover, some differences observable in the eyes. Much

* Hutchinson, "Med. Times and Gaz." 1868, p. 584.

† Seeligmüller, "Ueber Sympathicus-Affectionen bei Verletzung des Plexus brachialis," Berl. klin. Wochenschrift, 1870. No. 26.

less of the eyeball was seen through the palpebral fissure on the right side than on the left, in consequence of a marked difference in the vertical diameter of these fissures; the right pupil was at least one half smaller than the left, and was on some days contracted to the size of a pin's head, but still acting normally under the influence of light and shade. With respect to the colour and temperature of the skin, the two sides of the face were alike, but in the course of the disease a slight but perceptible atrophy of the right side of the face presented itself. The paralysis improved under the use of electricity; after three months the right pupil, too, was not so contracted as formerly, but with that exception the state of the pupils and of the atrophied parts remained unchanged. The second case was that of a man of 34 years, who was run over by a railway waggon, sustaining an injury to the left breast and shoulder, and fracture of the left forearm. Three months afterwards there were paralysis and emaciation of the left arm, with considerable disturbances of nutrition, and complete anæsthesia of the whole forearm. The left pupil was only half as widely dilated as the right, but quite sensitive to light, and the palpebral fissure on that side was only a very little shorter than that on the right. On instillation of atropine the contracted pupil became dilated almost as widely as possible, and in the following 48 hours contracted but slightly, though the paper prepared with Calabar bean was used. Galvanic irritation of the cervical sympathetic, though repeatedly tried, was followed only once by a transient dilatation of the pupil; whilst under this treatment, however, the left ear was usually warmer to the touch than the right, a sensation which was felt by the patient himself. He was a long time under observation, but the differences between the pupils remained unchanged.

The phenomena in the two last-mentioned cases correspond to a paralysed condition of the cervical sympathetic, but only in the sphere of the oculo-pupillary fibres, whilst the vasomotor fibres appear to be quite unaffected. Whether the trunk of the sympathetic itself, or the middle cervical ganglion, or the twigs passing from that ganglion to the brachial plexus, were injured, remains unknown.

Further, Bærwinkel,* in two cases of traumatic paralysis of the brachial plexus, (produced, in one instance, by fracture

* Bærwinkel, "Zur Pathologie des Sympathicus." *Deutsches Archiv. für klinische Medicin.* Bd. xiv., p. 545.

of the clavicle, in the other by a gunshot wound), observed paralysis of the oculo-pupillary sympathetic filaments, indicated by narrowing of the palpebral fissure, ptosis, and contraction of the pupil.

As far as we know there are no other published cases of the occurrence of paralysis of the sympathetic attendant on traumatic paralysis of the brachial plexus. Hutchinson errs in stating that this coincidental relation is *invariable*. We ourselves have, with this point in view, examined a considerable number of isolated, usually traumatic, cases of paralysis of the brachial plexus, both recent and of long standing, but without finding any trace of paralysis of the cervical sympathetic.

There may be disturbances of function in the cervical sympathetic which are not referable to any of the causes already mentioned, and these may appear in the domain of the sympathetic alone, and not in connection with any other complaint. In such cases also, as we formerly stated, the paralytic condition is more common than the irritative, and the oculo-pupillary fibres are more frequently and more persistently affected than the vasomotor fibres.

Several cases in point will be mentioned in the next section.

II.—UNILATERAL HYPERIDROSIS.

Among the symptoms following division of the sympathetic there is sometimes, as Cl. Bernard has stated with reference to horses, an abnormally profuse secretion of perspiration on the side of the head operated on. This has also been observed in the human subject in paralysis of the cervical sympathetic, and sometimes to such a degree that it was regarded as the principal symptom, and, as such, first attracted the patient's attention to his condition. Different cases of paralysis of the sympathetic, in which this was the predominating symptom, have been described under the special name Hyperidrosis or Ephidrosis unilaterialis. On the other hand, cases of unilateral secretion of perspiration have been recorded, some being confined to one side of the head, some extending over greater part of one side of the body, but in which other indications pointing to disorder of the sympathetic were entirely wanting. Setting aside some very old observations on this point, which have been brought together in a work by Nitzelnadel,* the above statements refer

* Nitzelnadel, "Ueber nervöse Hyperidrosis und Anidrosis," Jena, 1867, Inaug. Dissert.

specially to the more modern contributions of Meschede,* Berger,† Wiedemeister,‡ and others. Cases of Ephidrosis, however, which are with certainty known to be dependent on paralysis of the sympathetic, are rare. Verneuil and Ogle met with some, quoted in the former part of this work. Otto§ and Nitzelnadel|| mention one such, Bærwinkel¶ several. Kulz** describes two cases of Ephidrosis accompanying diabetes mellitus, though only the vasothermic, and not the oculo-pupillary, phenomena were present. Very aggravated cases, in which, besides the hyperidrosis, the other symptoms of paralysis of the sympathetic were unmistakably manifested, have been observed by Chvostek,†† Pokroffsky,‡‡ and ourselves. Our case was that of a man 44 years of age, who, after even very moderate exercise, perspired profusely on the left side of the face, and occasionally also on the left side of the throat and neck. Simultaneously with the breaking out of the perspiration the left side of the face and the left ear became red, and the temperature in the left external auditory meatus rose several tenths C. above that in the right. There was also considerable injection in the vessels of the left conjunctiva, while lachrymation was sometimes more easily excited in the left eye than in the right. The left pupil was constantly more dilated than the right, but responded to the stimulus of light. Accommodation remained normal, and there was no interference with nutrition. In the neighbourhood of the left cervical sympathetic there was some tenderness on pressure, perhaps the indication of a state of chronic inflammation of that nerve. It is worthy of note in this case that, while the vasomotor fibres were in a state of paralysis the pupillary fibres were in a condition of irritation.

Only two opportunities have presented themselves for investigating the nature of the pathological changes which take place in sympathetic hyperidrosis. In one case, Seguin¶¶ found no difference between the cervical sympathetic

* Meschede, "Virchow's Archiv." Bd. xliii., p. 139.

† Berger, "Virchow's Archiv," li., p. 427.

‡ Wiedemeister, "Virchow's Archiv." Bd. lii., p. 437.

§ Otto, "Archiv. für klin. Medicin." Band xi., Heft 6.

|| Nitzelnadel, L. c.

¶ Bærwinkel, "Archiv. für klin. Medicin," 1874. Bd. xiv., p. 550.

** Kulz, "Beiträge Zur Pathologie und Therapie des Diabetes," 1874, pp. 23 and 27.

†† Chvostek, "Wiener med. Wochenschrift," 1872. No. 19 and 20.

‡‡ Pokroffsky, "Berliner klin. Wochenschrift," 1875. No. 13.

¶¶ Seguin, "American Journal of Medical Sciences," Oct., 1872.

nerves of the right and left sides. The ganglionic cells seemed to be filled with an unusually abundant granular pigment, but this was equal on both sides. To the naked eye also they were alike. Ebstein* has recorded the case of a man, 60 years of age, who suffered from hyperidrosis of the left side, which appeared suddenly after a paroxysm of angina pectoris, and subsequently invariably accompanied each attack. On examination with the unaided eye no change was found in the sympathetic or its ganglia; but microscopic examination of thin sections of the ganglia of the *left* sympathetic revealed the presence of extremely varicose and dilated vessels, while on the right side no trace of such a structure was found. On the basis supplied by this discovery Ebstein explains hyperidrosis. He believes that these varicosities receive, at different times, a blood supply of varying amount. When the quantity is abnormally great, some of the sympathetic nerve elements must be temporarily compressed, and so paralysed—exactly as, in very vascular new formations in the brain, various paralytic phenomena occur in different parts of the nervous system, all of which disappear when the quantity of blood in circulation is reduced.

III.—HEMICRANIA.

Our conception of the proper nosological position of Hemicrania was, till lately, very obscure. Old authors (as Wepper, Tissot) have identified the disease with Prosopalgia, especially that form occurring as supra-orbital neuralgia; and Schönlein, who classifies hemicrania among the neuroses of the genital system, calling it "*Hysteria Cephalica*," places the seat of pain in the ramifications of the frontal and temporal nerves. Even at the present time there are not wanting pathologists (Lebert, Stokes, Anstie, Clifford Albutt) who look upon hemicrania as a simple neuralgia, affecting the first division of the trigeminus. Believing in this doctrine many men have somewhat arbitrarily distinguished between various forms of hemicrania, according to the presumed or actual causes giving rise to it. Thus, Sauvages speaks of ten varieties of hemicrania, and Pelletan of "*Migraine stomacale, irienne, utérine, pléthorique*." Monneret and Fleury recognise "*Migraine idiopathique*" and "*sympathique*," a classification which is adopted also by Valleix. An important step was taken by Romberg when

* Ebstein, "*Virchow's Archiv.*," 1875. Bd. lxi., p. 435.

he associated hemicrania with the "Hyperæsthesiæ of the brain," with painful affections of the brain, thus defining it sharply from the peripheral neuralgias, calling it "Neuralgia cerebialis." Later pathologists took the same view; thus Leubuscher calls hemicrania *the* neuralgia of the brain. The fact that we are able to localise the seat of the pain in the brain, or in the parts of it endowed with sensation, does not much advance our knowledge of the pathogeny of the disease, and Romberg's conception of hemicrania as one of the cerebral neuroses is open to the same objection. Romberg was, as it appears, specially led to that conclusion by the sympathetic connections existing between the trigeminus and the nerves of special sense, and by the favourable effect produced by physical and mental exercise. But Hasse has remarked, with justice, that deciding according to the analogies offered by other neuralgias, the sympathetic connection of several cranial nerves, and the reflex phenomena resulting therefrom, give no ground for assuming the ramifications of the trigeminus within the cranium as the seat of the disease.

Sixteen years ago Du Bois-Reymond,* from personal observation, inferred that his migraine was caused by *tetanus of the muscular coat of the vessels on the affected side of the head*, or, in other words, *tetanus in the region supplied by the cervical part of the (right) sympathetic nerve*. He found that during the attack the temporal artery of the painful side was hard and cord-like to the touch, while that on the left side was in its normal state. The face was pale and sunken; the right eye small and injected. The pain was increased by everything which raised the blood-pressure in the head (as stooping, coughing, &c.), this increase being synchronous with the pulse in the temporal artery. Towards the end of the attack the right ear became warm and red. These phenomena, the state of the temporal artery, the bloodlessness of the face, the sunken appearance of the right eye, show that the muscular coat of the vessels of the affected side of the head was persistently contracted. On removal of the cause which produces this condition of tonic spasm, relaxation follows the overaction of the unstriped muscular fibres, and the walls of the vessels yield more than usually to the lateral pressure. This secondary relaxation explains the congestion of the conjunctiva, and the redness and in-

* Du Bois-Reymond, "Zur Kenntniss der Hemikranie, Archiv. für Anat. und Phys.," 1860, p. 461-468.

creased temperature of the ear, which occur when the violence of the attack begins to subside. The vomiting and the flashes of light before the eyes which frequently accompany hemicrania are caused by sudden changes in the intracerebral blood-pressure; and these variations obviously correspond to the irregular contraction and relaxation of the unstriated muscular coat of the vessels.

Such a pre-supposed tonic vascular spasm of one side of the head can, as we know from physiological facts, have its origin only in the sympathetic nerve of the same side, or in the medullary centre of the sympathetic fibres involved; that is, in the corresponding half of the regio cilio-spinalis of the spinal cord. Hemicrania is thus to be regarded, not as a neuralgia of peripheral nerves or of the brain, and generally not as a primary cerebral disease, but as an affection of the cervical sympathetic nerve, or of a certain part of the spinal cord. This apparently rash assertion receives strong support from a further observation by du Bois-Reymond. He noticed, in the course of the attack, *a dilatation of the pupil on the affected side*. A medical visitor confirmed this observation: and the more shaded the eyes were, the more decided was the difference in the size of the pupils, exactly as in tetanus of the cervical part of the sympathetic. Latterly, during and after the attack, the spinous processes corresponding to the regio cilio-spinalis were painful to pressure.

Whether, at the end of the paroxysm, there was decrease in the size of the pupil in conjunction with the elevation of temperature and the redness, was not stated; but in several instances, otherwise identical in type with that quoted, we have observed a decided contraction of the pupil on the affected side of the head as the attack was passing off. Brunner* remarked in his own case, besides the symptoms mentioned by du Bois-Reymond, pain on pressure in the neighbourhood of the upper, and sometimes also of the middle cervical ganglion. This tenderness disappeared slowly, usually on the following day, and palpitation of the heart and acceleration of the pulse were frequently observed at the close of an attack.

Brunner noticed the same symptoms also in the case of his mother. We ourselves and some other authors have seen, in several analogous cases of migraine, a more or less considerable increase in the quantity and tenacity of the saliva.

* Brunner, "Zur Casuistik der Pathologie des Sympathicus." Petersburger med. Zeitschrift, N. T. Bd. ii., 1871, p. 260.

In one case, Berger* found that two pounds of tough saliva had been discharged. This symptom, also, is to be classified with those already mentioned, since secretory fibres for the salivary glands are included in the cervical sympathetic, and irritation of these in animals produces similar effects. The question arises, in what relation the tetanus of the region supplied by the right sympathetic stands to the hemicranial pain, whether it is only a concomitant symptom, or the cause of the migraine, that is, of the paroxysmal attacks of pain. With regard to this, du Bois-Reymond has suggested that the state of tonic spasm of the unstriated muscles of the vessels may itself be that which causes the pain: just as it is felt in striped muscular fibre, in cramp of the calf of the leg, and in tetanus; or in unstriated muscular fibre, in the uterus during labour-pains, in the intestines during an attack of colic, and in the skin during rigors. Probably this pain comes from pressure on the nerves of sensation distributed within the muscular tissue; this pressure, and consequently the pain, will be augmented when the tetanic muscles are more strongly exerted, as, for instance, in the case of cramp in the calf when the muscles are extended, either by means of the antagonistic muscles, or by the weight of the body, the ball of the foot being supported. This also is produced in tetanus of the muscular coat of the vessels when the lateral blood-pressure is increased. Thus a reasonable explanation is found for the observation that pain is increased along with the blood-pressure, and synchronously with the pulsations of the temporal artery.

Besides du Bois-Reymond's explanation of the pain, another one appears to us worthy of mention, being perhaps more probable and less forced. In the variations in the flow of arterial blood, especially in the temporary anæmia of the affected side of the head, a shock may be given which irritates the sensory nerves of the head, whether those in the skin, the pericranium, the membranes of the brain, the sensitive parts of the brain itself, or in all these at once, and thus causes the hemicranial pain. Sensory nerves are brought into a state of great excitement by changes in the diameter of the accompanying and surrounding bloodvessels, especially when these changes occur with a certain degree of suddenness. This condition is met with in very different cases of neuralgia, as in facial neuralgia and sciatica, &c.

* Berger, "Zur Pathogenese der Hemicranie." *Virchow's Archiv*, lix., Heft 3 and 4, 1874.

Those neuralgias, also, which usually follow herpes zoster—which occur most commonly in the body, but sometimes in the face and extremities—are very probably to be referred to this source. Anomalies of the circulation generally, and anæmia specially, have been long ago recognised as important causes of neuralgic affections in different parts of the body. The aggravation of the hemicranial pain in stooping, coughing, &c., and the peculiar effect of compressing the carotid, originate in this way. In many cases of migraine the pain ceases on compressing the carotid on the painful side, but is increased by compression of the carotid on the sound side. In one case, however, we observed the opposite—that the pain became decidedly worse on exercising pressure on the carotid of the same (the right) side, while compression on the left side at once alleviated it. This case strikingly demonstrates the good effects of local anæmia. Probably the direct cause of migraine is to be found in the local alterations in the circulation, while the state of the muscular coat of the vessels, being the most common cause of these variations, can be regarded as playing only a secondary part in the origination of the affection. The inequality and inconstancy of the pupillary and vasomotor phenomena are strongly in favour of this view.

The objection raised by Brown-Séquard* and Althann† against du Bois-Reymond's theory, that, according to the experiments of Kussmaul and Tenner, the occurrence of epileptic convulsions should be expected in unilateral vascular tetanus in the brain, is not defensible, since it is proved, both by the formerly-quoted experiments of Fischer, and by those performed by ourselves, that in unilateral experimental irritation of the cervical sympathetic convulsions do not occur, but always contraction of the vessels and lowering of the temperature on one side. The same symptoms occur also in pathological cases of unquestionable (mechanical) irritation of the sympathetic in men, as has already been in part described in the foregoing section. That there is a certain genetic relation existing between migraine and epilepsy is beyond a doubt; suffice it to state that in hereditarily and constitutionally predisposed epileptics migraine is one of the most frequent accompanying symptoms, both in early life

* Brown-Séquard, "De l'hémicranie ou migraine, par le Dr. du Bois-Reymond," *Journal de Phys.* 1861.

† Althann, "Beiträge zur Physiologie und Pathologie der Circulation." *Dorpat*, 1871.

and afterwards with fully-developed epilepsy, and that in families prone to be affected with constitutional neurophatic complaints some members often suffer from migraine, others from epilepsy, and various affections belonging to the same group.

Du Bois-Reymond has remarked that by no means all cases of migraine present the above-mentioned symptoms during the attack; that especially the difference in the size of the pupils in many otherwise very pronounced cases of periodic unilateral headache is not observed. He has therefore proposed the name *Hemicrania Sympathico-tonica* for those cases resembling his own, in which one may assume the existence of tetanus in the cervical part of the sympathetic as a patho-genetic force. More recently, Möllendorff,* apparently without any knowledge of du Bois-Reymond's paper, has set up a theory of migraine which really amounts to this—that hemicrania is due to a unilateral *loss* of energy in the vaso-motor nerves governing the carotid artery, whereby the vessels are *relaxed*, and permit of an increased flow of arterial blood towards the head. In the case recorded by Möllendorff the rate of the pulse was reduced to 56—48 per minute, the radial arteries were small and contracted, and the pulse in the carotid and temporal arteries of the painful side was soft and wavy. Compression of the carotid of the affected side during the attack instantaneously dissipated the pain, which returned with the first beat of the pulse on slackening the pressure. On the other hand, the pain was aggravated by pressure on the carotid of the other (sound) side.

Ophthalmoscopic examination of the eye on the painful side, in a patient suffering from migraine, showed during the attack dilatation of the central vessels, the art. and vena centralis retinae, the latter being knotted and convoluted, and of much darker colour than usual; the choroidal vessels also were enlarged, so that the fundus of the eye was of a bright scarlet colour instead of its usual dark-brownish red. Sometimes there was considerable injection of the episcleral vessels as far forward as the edge of the cornea, disappearing on subsidence of the attack. The fundus of the other eye was normal, the art. and vena centralis having their usual appearance. It is, unfortunately, not stated at what stage of the attack the examination was made, though probably it was towards the end. In a similar case Berger and H. Cohn

* Möllendorff, "Ueber Hemicranie," Virchow's "Archiv. für path. Anat." Bd. xli, p. 385-395.

found the fundus of the eye normal. In several analogous cases of migraine we ourselves observed a more or less marked *contraction of the pupil*, sometimes a diminution in the size of the palpebral fissures, retraction of the globe of the eye, partial ptosis in, and difficulty of moving, the upper lid. The ear on the same side was red and hot, and the temperature in the external auditory meatus was raised $0.2-0.4^{\circ}$ C. The secretions of the skin were increased, and sometimes ephidrosis unilateralis occurred.

It thus follows that there are cases of hemicrania which are entirely opposed in character to that of du Bois-Reymond—that is, in which the striking symptoms are not those of *spasm* in the vessels, of arterial tetanus in the parts supplied by the cervical sympathetic, but those of *relaxation* of the vessels, of arterial hyperæmia caused by a loss of energy in the vasomotor nerves. These are cases that one might designate *Hemicrania Neuroparalytica*, or *angioparalytica*, as opposed to du Bois-Reymond's *H. Sympathico-tonica*. In such circumstances the occurrence of the pain can certainly not be explained in the same way as in the former class of cases.

Our interpretation of the phenomena, however, is applicable here, as the temporary increase of the blood-pressure, the greater quantity of blood in the small arteries and veins, gives rise, by irritation and compression of the nerve elements, to the pathognomonic symptoms of hemicrania in exactly the same way as in the opposite case—arterial anæmia and decreased blood-supply from spasm in the vessels. We know, further, from other sources, that increased or diminished blood-supply, anæmia or hyperæmia, agree closely in their action on the brain; that, for instance, epileptic seizures take place both in anæmia of the brain (according to the Kussmaul-Tenner experiments) and in hyperæmia of the brain (through retardation of the return of venous blood by closure of the vena cava superior*), and that the effect on the action of the heart and the frequency of the pulse is quite analogous in both conditions. †

The lowering of the rate of the pulse, which Möllendorff does not explain, has been noticed by Landois in hyperæmia of the brain and medulla oblongata, produced artificially by compressing the superior vena cava; he has observed it also after extirpating both cervical sympathetic

* Hermann und Escher, Pflüger's "Archiv. für Physiologie," 1870, p. 3.

† Landois, "Centralblatt für die med. Wissensch.," 1865. No. 44; 1867, No. 10.

nerves, but not when the spinal marrow had been previously destroyed or the vagi divided. This decrease in the frequency of the pulse, which, in congestion of the brain, may proceed to arrest of the heart's action, and may be complicated with epilepsy, is dependent on a *direct*, not a reflex, *irritation of the medulla oblongata and of the vagi*; division of the latter, whilst there is hyperæmic retardation of the pulse, is at once followed by acceleration of the pulse.

Since the centres for most of the vaso-motor nerves of the body are situate in the medulla oblongata, irritation of that important part of the nervous system furnishes a full explanation of the fact that the radial arteries are small and contracted in hemicrania, of the occurrence of icy coldness of the hands and feet and cold shiverings over the whole body, and of the suppression of perspiration during the paroxysm. The last-mentioned symptom, however, is often absent on the affected side of the head. This contraction of the peripheral arteries is followed by dilatation, by secondary relaxation. It may be this which gives rise to the augmentation of the secretion of saliva and urine occurring towards the end of an attack, and also to the swelling of the liver and hypersecretion of bile, to the gradually developed plethora of the abdominal organs, the tendency to bronchial catarrh and emphysema of the lungs, which, Möllendorff alleges, eventually appears in persons affected with hemicrania.

The views advanced by du Bois-Reymond and Möllendorff appear to us to be of some importance in the *therapeutics of hemicrania* but in a way quite different to that claimed for them by these authors. Du Bois-Reymond merely hints that in the form described by him remedial measures should be directed specially to the regio ciliospinalis. Möllendorff says absolutely nothing concerning local treatment; and yet it is specially to local measures, as opposed to the hitherto fruitless general treatment and empiricism, that we should look for most benefit. It is, further, on local experimental evidence that we found the doctrine that migraine is a periodically recurring neurose of the vessels of the head, an affection of the cervical sympathetic, or of the central origin of the vasomotor filaments of the vessels of the head—the regio ciliospinalis of the spinal cord. Bernatzik* ascribed the well-known effects of caffeine and quinine in cases of migraine, in the stage of relaxation which follows the primary spasm

* Bernatzik, "Wiener med. Presse," 1867. No. 28.

of the vessels, to decrease in the quantity of blood in the vessels of the brain; he represented the operation of these remedies as depending really on irritation of the vasomotor nerves, on increase of the arterial tone. It is possible that the above-mentioned remedies may be of special use in those cases of migraine that we distinguished as neuro-paralytic or angio-paralytic, in which, during the attack, the most prominent symptoms are those of relaxation of the vessels and of arterial hyperæmia.

We ourselves met with a case clearly belonging to the last category, in which quinine had a rapid and surprising effect. The case was that of a boy, eight years of age, who suffered from a daily attack of hemicrania in the left side of the head; it began usually about midnight, and lasted till the forenoon of the following day, gradually declining in intensity. The boy had previously recovered from what was said to be "*Scarlatina sine exanthemate*," and was, besides, affected with torticollis spasticus from contraction of the left sternomastoid muscle; no further etiological indications were found. Both sides of the face were usually the same in colour; at the height of the attack, however, the left side of the face and the left ear were of a deep red colour and (especially the ear) warm to the touch, while, on subsidence of the pain, this relation often appeared reversed. The upper part of the face, the forehead and orbital region, took no decided part in these changes, and no difference in the size of the pupils was observable. On the exhibition of 0·5 gramme of sulphate of quinine his usual nightly seizure did not occur, but returned next night as before. A second similar dose was followed, first by a slight relapse, and then by complete recovery, which has lasted till the present time, five months from the appearance of the last attack.

In accordance with the preceding view, another drug recommends itself for further trial in cases of the angio-paralytic variety of migraine—the *extractum secalis cornuti aquosum*—which, we know, causes contraction of the blood-vessels, an action which, according to Wernich, Holmes, Vogt, and others, probably takes place partly through the vasomotor nervous centra in the medulla oblongata. We ourselves, Berger, and others have used this remedy, which has already been highly praised by Woakes,† in cases of hemicrania that were decidedly of an angio-paralytic

† "*British Med. Journal*," 1868, vol. ii., p. 360.

character. We administered it both internally (0·6-0·9 gramme daily, in the form of pill) and subcutaneously, with very considerable relief to the symptoms.

Another remedy, lately employed for the first time, appears destined to play an important part in the treatment of cases of the sympathico-tonic variety of migraine—at least, as a palliative. This is *nitrite of amyl*, introduced by Guthrie in 1859. The indication for its use is based on the fact that it dilates the bloodvessels, whether by directly acting on their contractile elements (Richardson, Lauder Brunton, Wood, Pick), or by paralysing the vasomotor nervous system (Bernheim, Filehne), is still undecided. When inhaled, it provokes coughing, and occasions intense redness of the face, a sensation of heat in the face and head, and injection of the conjunctiva; it quickens the pulse by 20-30 beats per minute, lessening the tension in the radial artery. Further inhalation may even cause syncope. O. Berger* used it in a case of migraine of the sympathico-tonic type with an almost instantaneously successful result. The patient, an unmarried lady of 24 years of age, suffered many years from a migraine of the left side of the head, occurring regularly at the otherwise normal catamenial periods, and occasionally, also, in the intervals. The attacks usually began in the morning, reached their greatest intensity about dinner-time, and lasted with still considerable severity till late in the evening. There was also a feeling of exhaustion on the following day. During the attack the left side of the face was pale and sunken, the temporal artery very prominent, hard to the touch, and pulsating so strongly as to be almost audible to the patient. There were frequent shiverings over the whole body. There was no very evident reddening of the face or ear of the affected side, and the appearance was pretty much the same throughout the whole day. On the other hand, she noticed that now and then, in the intervals when she had no pain, sometimes without any assignable cause, and occasionally when emotionally affected, the left side of the face and the left ear became intensely red, contrasting with the colour of the right side. On inhaling five drops of the nitrite, the pain was as if charmed away. She first felt as if the blood were rising to her face, and had a certain confused sensation in the head, but the violent, boring pain of the migraine itself had disappeared. Vomiting did not occur. She was

* Berger, "Das Amylnitrit, ein neues Palliativmittel bei Hemikranie," *Berliner klin. Wochenschrift*, 1867. No. 2.

able to take dinner, but towards evening had to retire to rest, as she was in a not unpleasant state resembling intoxication. At this time she was strikingly pale, but showed no further evil after-effect, and next day was perfectly well. Berger directed her to use only three drops on being again attacked, to repeat the inhalation in a quarter of an hour if she thus obtained no good effect, and even to increase the dose to 6-8 drops.

Besides Berger, Vogel and Holst* extol the virtues of nitrite of amyl in migraine. Holst made some experiments on himself and on five patients who presented typical symptoms of spasm in the muscles of the vessels. Inhalation of 3-5 drops produced a sensation as if the blood were rushing to the head, the face became red, and if the inhalation were not suspended, momentary insensibility occurred. At the same instant, however, the pain in the head vanished. In the case of Holst himself, and in that of one patient, motion caused the pain to return in a few minutes with all its former violence; two other patients, who remained quiet, had a relapse in an hour; another, who kept perfectly still, was not only relieved of that paroxysm, but the next remained absent longer than usual, and was also removed by the nitrite of amyl.

Holst also observed in himself that in well-marked attacks of migraine drinking freely of anything warm gave instant relief when a general perspiration broke out. This he explains by relaxation of the vascular system, which was previously in a state of tonic contraction.

The beneficial influence of the inhalation of carbonic acid, much lauded by A. Mayer,† may be traced to the fact that this gas, by paralysing the vasomotor nerves, removes a temporary state of spasm.

The constant galvanic current is another remedial measure that may lay claim to great importance in the different forms of migraine. It appears to be better adapted for the treatment of this affection than any other means we know of, inasmuch as we can exercise a real and powerful influence, strictly localised, and exactly regulated as to quantity and quality, on the cervical sympathetic nerve and the upper part of the spinal

* Holst, "Ueber das Wesen der Hemikranie und ihre electro-therapeutische Behandlung nach der polarne Methode." *Dorpater med. Zeitschrift*, 1871. Bd. ii, p. 261-228.

† "Wiener med. Presse," 1865, No. 46, p. 1123.

cord. Benedikt,* Trommhold,† Fieber,‡ M. Rosenthal,§ Althaus,|| and others, have written on this subject; it was Holst,¶ however, who first introduced the really methodical and rational use of the constant current, on Brunner's polar system, in the different forms of migraine. His practice is to put one electrode on the cervical part of the sympathetic, at the inner edge of the sterno-mastoid and in contact with a considerable surface, and to establish communication with the other electrode, which is held in the palm of the hand. In hemicrania sympathico-tonica the anode is placed on the sympathetic, and a battery of 10-15 elements used; the circuit is suddenly closed, and the current, after being passed 2-3 minutes, is gradually reduced in strength. This is copied from Brunner's mode of proceeding in irritable conditions of the auditory nerve. In hemicrania neuro-paralytica the cathode is applied to the sympathetic, and the circuit is not simply closed, but is made to produce a more powerful effect by repeated interruptions or by reversing the current. The first-mentioned method, which directly diminishes irritability, was most often resorted to by Holst, especially in cases in which the condition of the muscular coat of the vessels was doubtful, as he regards an abnormal irritability of the vasomotor nervous system of certain parts of the head as the primary cause in every hemicrania, even in those which are characterised secondarily by a paralytic state, and he believes that by lessening this abnormal irritability the tendency to secondary relaxation of the walls of the vessels is probably overcome. Holst's own results, in about thirty cases, are, on the whole, in favour of this method of treatment. For details we must refer to the original work, only remarking that in all cases in which there is spasm of the vessels there is considerable improvement a few seconds after passing the current, the anode being placed on the sympathetic, and that this is often conjoined with a sensation of warmth in the head and heat and redness in the ears.

In one very obstinate case of hemicrania of the neuro-paralytic type (the face being flushed and hot during the

* Benedikt, "Elektrotherapie." Wien, 1868.

† Trommhold, "Die Migraine und ihre Heilung durch Electricität." Pest, 1868.

‡ Fieber, "Compendium der Elektrotherapie." Wien, 1869, p. 120.

§ M. Rosenthal, "Handbuch der Diagnostik und Therapie der Nervenkrankheiten," Erlangen, 1870.

|| Althaus, "Treatise on Medical Electricity," 3rd edition. London, 1873.

¶ Holst, l.c., p. 275.

paroxysm), which never entirely intermitted, but took the form rather of a series of exacerbations and remissions, occurring in the person of a girl of 17, treatment by the interrupted galvanic current, the cathode being applied to the sympathetic, gave marked relief; though this did not last long, perseverance in the use of the same means for weeks was thus far successful that the painless intervals gradually became longer than the attacks, and subsequent treatment by reversing the current was followed by still more decided improvement.

IV.—GLAUCOMA. NEUORETINITIS. OPHTHALMIA. NEUROPARALYTICA.

In this section we discuss the imperfectly known connection which exists between certain diseases of the eye on the one hand, and functional disorders in the domain of the cervical sympathetic on the other.

We will first consider:—

Glaucoma.—This disease consists, as is proved in v. Gräfe's celebrated works, of *an increase of the intraocular pressure*. Inflammatory changes (choroiditis and disorders of nutrition in the vitreous body) may, as a rule, be regarded as the causes of this increased pressure. Remak, however, expressed the opinion, unaccompanied by any very convincing arguments, that glaucoma may have its origin in primary disease of the spinal cord. Adamük and Wegner have observed, in their investigations on the dependence of intraocular pressure on the agency of the cervical sympathetic, that the latter has a great influence on the glaucomatous process. Adamük believes that the ultimate cause of glaucoma is not increased pressure, but obstruction to the return of venous blood, produced by loss of elasticity in the sclera, which, again, is the result of inflammation; and that the sympathetic is only thus far involved—that irritation of it is followed by contraction of the arteries and overloading of the veins in the fundus of the eye, and by increase of tension.

Wegner, who examined two cases of glaucoma simplex, accompanied by neuralgia of the trigeminus, has come to the conclusion that the sympathetic vascular nerves may, in three ways, take part in the production of glaucoma: they are either directly concerned in the inflammatory process, or irritated by pressure, or stimulated, reflexively, by the sensory trigeminus nerve; he referred to the latter cause the two cases mentioned.

Hippel and Grünhagen reject this explanation, being of opinion that both acute glaucoma and glaucoma simplex, without any inflammatory symptoms, proceed directly from the trigeminus, irritation of which occasions, according to them, an increase of the intraocular pressure. They state that the less the vascular tone (which is dependent on the sympathetic), the more readily does this increased pressure appear, as tonicity counteracts the tendency to such increase. (We think ourselves justified in omitting to mention here the numerous theories of glaucoma not directly connected with the sympathetic.)

Illustrative clinical instances of the disease are very rare. In various cases of mechanical irritation, or compression of the cervical sympathetic, we noticed no glaucomatous phenomena; in one case, formerly described (irritation from goitre on the right side), there was a considerable degree of tension of the globe, which, however, never assumed any other than a physiological character; after being some months under observation no arterial pulse or excavation of the papilla appeared.

Horner,* Bærwinkel,† and Schmidt-Rimpler,‡ have, in some cases in which there was presumably paralysis of the sympathetic filaments, observed increased tension in the eye. According to Schmidt-Rimpler§ the existence of an influence exerted by the sympathetic on increase of intraocular pressure, and thus on the occurrence of the glaucomatous process, is not to be denied; nevertheless, this state is found more seldom, and to a slighter degree, than that resulting from disease in the trigeminus.

Neuroretinitis.—Benedikt|| teaches that the sympathetic system often plays an important part in causing those *intracranial, limited regional diseases* ("Heerderkrankungen") which lead to secondary affections of the optic nerve, or retina.

The forms of disease under consideration are those described as Neuritis, Atrophia descendens, and "engorged papilla." Benedikt declares that it is an unsatisfactory explanation of the phenomena to assert the existence of a "neurore-

* Horner, "Ueber eine Form von Ptosis." *Klin. Monatsbl. für Augenheilkunde*. 1869, vii., p. 193; Nicati, "La paralysie du nerf sympathique cervical," 1873.

† Bærwinkel, "Archiv. für klinische Medicin." 1874, Bd. xiv., p. 549.

‡ Schmidt-Rimpler, "Klin Monatsbl. für Augenheilkunde," xii., p. 398.

§ "Handbuch der Ophthalmologie," Bd. v. (1875), p. 98 and 99 (Capitel "Glaucom").

|| Benedikt, "Elektrotherapie." Wien. 1868, p. 253 ff.

tinitis," and an engorgement of the retinal vessels (v. Gräfe)* arising from increase of intracranial pressure. He advances the theory that in the local diseases within the cranium one has often to do with antecedent or accompanying neuroses of the sympathetic vasomotor fibres; that the symptomatic neuroretinitis, in most cases, depends on a morbid condition of the sympathetic, which is, further, a symptom observed in various other cerebral disorders. In the same way also he endeavours to explain the occurrence of other symptomatic phenomena—such as functional derangements of the auditory nerve in diseases of the brain, secondary affections in parts of the brain which are situated at a distance from the original limited diseased area (such as the sympathy shown in the cortical substance of the brain in cases of tumour in the pons), and hydrocephalus in those cases in which the idea of the extension of the morbid action to the walls of the ventricles by continuity of tissue is out of the question. These views, so ingeniously worked out by Benedikt, may, doubtless, greatly help us in understanding the causation of many secondary disturbances of the circulation in local cerebral diseases; as regards specially his conception of "neuroretinitis," however, as a secondary, sympathetic neurose, it seems to us to be very deficient in positive, confirmatory evidence—at least his presumed sensibility of the sympathetic in the neck, and the therapeutic success following galvanization of the sympathetic in chronic diseases of the brain, are of little value in this respect.

H. Schmidt's† experiments have lately demonstrated a direct communication between the subarachnoid space and the lamina cribrosa, and supply a good explanation of the occurrence of the engorged papilla and simple white atrophy when the intracranial pressure is increased. The former difficulties in the way of giving a reason for engorgement of the retinal vessels‡ are thus removed; while, on the other hand, in most cases of engorged papilla and simple white atrophy, they render superfluous Benedikt's supposition that the neuroretinitis is a secondary sympathetic neurose.

* v. Gräfe, "Ueber Neuroretinitis," *Archiv für Ophthalmologie*, 1866 Bd. xii., p. 114.

† H. Schmidt, "Zur Entstehung der Staunungspapille bei Hirnleiden." *Archiv für Ophthalmologie*, 1869. Bd. xv., p. 193.

‡ Seseman (*Archiv für Anat und Phys* 1869, p. 154) showed that compression of the cavernous sinus is not followed, as v. Gräfe believed, by great engorgement in the retinal veins, as the return of blood, by direct communication with the superficial veins of the face, is quite free enough.

With respect to the influence exercised by the cervical sympathetic on the occurrence of

Ophthalmia neuromyolytica (an influence the existence of which is not improbable, bearing in mind the fact that vasomotor fibres pass from the sympathetic to the trigeminus) we quote an observation made by Walther,* and mentioned by Henle.† Here, after division of the sympathetic in extirpating an aneurism of the carotid, ophthalmia appeared; the disturbance of the circulation, however, following such an operation, may, perhaps, alone suffice to account for the ophthalmia. According to Sinitzin's formerly-quoted (somewhat untrustworthy) experiments, division of the sympathetic should rather ward off ophthalmia, as it lessens the irritability of the globe of the eye; he states, nevertheless, that ligature of the carotid on the corresponding side equalises the difference in irritability, and does away with the consequences of extirpating the sympathetic.

V.—PROGRESSIVE FACIAL HEMIATROPHY.

In a former part of this paper we mentioned some cases in which mechanical injury of the cervical sympathetic was followed by emaciation of the corresponding side of the face. There is a well-known disease which is characterised by atrophy of one side of the face, usually first attacking the superficial soft parts, and then the deeper tissues—a disease which has been called *Prosopodysmorphia* by Bergson, *Neurotic facial atrophy* by Samuel and Bäerwinkel, "*Aplasie lamineuse progressive*" by Lande, and *progressive facial hemiatrophy* by others. Romberg and Bergson, and, at a later date, Samuel, regarded it as connected with the nerves of nutrition; while Stilling believed it to be the result of disturbed function in the vasomotor nerves, especially in those filaments included in the trigeminus, and destined for the vessels of the head. Stilling's view, that there is diminished reflexion from the sensory nerves of the vessels to the corresponding vasomotor nerves, is somewhat strained, and it might easily be shown that the vasomotor nerves of the face take a *direct* share in the morbid action, especially those which form part of the trigeminus, as in the cases recorded by Axmann, Hüter, and others.

Bäerwinkel endeavours to show that in the cases observed by him, in which there was atrophy in the region supplied by

* "Gräfe's und Walther's Journal," xxix 1840, p. 549.

† "Nervenlehre," p. 570.

the infraorbital nerve, we must admit the existence of disease in the sphenopalatine ganglion. But Lande has lately denied that the disease has a neurotic origin, and maintains that we have to do with a genuine and primary atrophy of the fatty tissues, that the elastic tissue remains unaffected, its retraction causing the falling in of the other soft parts and contraction of the capillary vessels, the latter leading to further disturbances of nutrition. Nevertheless, many considerations render this hypothesis doubtful; it would not exclude, as Lande himself admits, the co-operation of nerves of nutrition (the existence of which he erroneously regards as demonstrated) in the production of the disease, since the primary shrinking of the fatty cellular tissue might possibly itself depend on a lesion of the vasomotor nerves of nutrition.

We will not here further discuss these relations, but only state that till very lately we were in possession of so few facts that would justify us in believing the disease to be in any way related to the sympathetic, that attention had been turned rather to the vasomotor nerves of nutrition included in the trigeminus. Since, however, these fibres, before joining the trigeminus, are for the most part contained in the cervical sympathetic, there is still some possibility of the latter being involved, and the possibility is indirectly rendered more probable by the circumstance that slight atrophy of one side of the face is observed in some cases of injury of the sympathetic in the neck. Brunner* has lately recorded one striking case of unilateral atrophy of the face, which he believes points to a diseased state, a condition of permanent irritation, of the cervical sympathetic. The case occurred in the person of a Jewish lady, 27 years of age, who, during pregnancy, had an attack of convulsions with loss of consciousness, and afterwards repeated epileptic seizures. For a long time these attacks followed regularly on each faradisation of the facial muscles, and were ultimately associated with difficulty of breathing and palpitation. In the course of four years an atrophy of the left side of the face was gradually developed, the hair of the head and the eyelashes became grey, and several yellow and white spots appeared on the skin, which afterwards assumed a yellowish brown, or brown colour. There was also a feeling of pressure and cold in the left eye, pain in the whole left side of the face, and in the jaw and throat; violent pain in the neck and chest, as

* Brunner, "Zur Casuistik der Pathologie des Sympathicus" *Petersburger med. Zeitschrift*, N. F. Bd. ii., 1871, p. 260.

far down as the region of the stomach, the latter sensations presumably only on the left side. The frontal and temporal muscles were found to have almost quite disappeared, and the zygomatici and the other muscles of the angle of the mouth, of the nose, and lips, more or less atrophied, and some of them at the same time contracted; their electro-muscular contractility was intact. The external part of the left ear was, on the whole, much thinner, smaller, and cooler than that of the right. The left eye appeared larger than the right, *the palpebral fissure wider, and the eyeball more prominent, the pupil more dilated and sluggish in its action.* The conjunctiva was rather pale, its vessels being sparingly filled with blood; the secretion of tears and mucus was diminished. The skin of the whole face was very thin and dry, and the subcutaneous fatty cellular tissue almost entirely absent. One side of the face was always paler, even when reddened by heat, cold, or mental changes; it took almost no part in perspiration, only the nasal fold being somewhat moist. The temperature in the right side of the mouth was $\frac{1}{5}^{\circ}$ C. higher than in the left, and in the right auditory meatus about 1° higher than in the left. There was pain on pressure on the left ganglion cervicale supremum, but none on the right; pressure on the ganglion cervicale medium on both sides produced slight pain. The heart sounds were clear, but irregularly accentuated, the same being the case in the carotid sounds; frequency of pulse variable, 86—100 in the minute.

Brunner thinks that the symptoms in this case correspond to the state experimentally produced in animals by galvanization of the divided cervical sympathetic, or its ganglion supremum. The dilatation of the pupil, its sluggish action, the widening of the palpebral fissure, the exophthalmos, the scanty secretion of tears and mucus, the feeling of tension in the eye (as in glaucoma), the lowering of temperature on the whole left side of the face, the absence of perspiration, &c., are to be explained in this way; and the formerly mentioned cases of Ogle and others show that very similar symptoms accompany pathological irritation of the sympathetic. Brunner concludes that a more lasting, or constant irritation of the sympathetic (perhaps of inflammatory character, or caused by a tumour) gave rise to a persistent state of spasm in the blood vessels, and to the train of symptoms already described in his case. The palpitation of the heart is also to be referred to deficient innerva-

tion by the sympathetic; and the same may be said of the epileptic seizures which follow spasm of the vessels of the medulla oblongata or the basilar parts of the brain, and, possibly also, according to Benedikt and Meynert, of the left hippocampus major. Brunner further believes he has proved that the trigemini and facial nerves have no connection with it, and that the slight pains in the atrophied side of the face are perhaps to be explained by muscular sensation, or disturbance of nutrition. In accordance with his view of the case, Brunner cautiously employed the galvanic current, long continued, of weak tension, of few elements, and as *constant* as possible, confining its action to the two upper cervical ganglia. Galvanization practised in that manner was at once followed by quieting of the heart's action and slight dilatation of the pupil, while the affected side of the face became red and covered by a profuse perspiration. Brunner reckons on obtaining successful results from this method of treatment on the supposition that, as is probable from the persistence of the symptoms of irritation and the pain on pressure on the upper ganglion, we have to do with an irritation or chronic inflammation of the latter, not with a malignant degeneration.

In one case, observed by us, galvanization produced reddening of the affected side, which lasted some hours, yet, after having used the constant current some months, we could not boast of any permanent good result. In this case, however, we had no decided symptoms of an affection of the sympathetic.

VI.—PROGRESSIVE MUSCULAR ATROPHY.

Although Cruveilhier and Aran are usually regarded as the original discoverers of progressive muscular atrophy, and, to a certain extent, with justice, the celebrated work of Sir Charles Bell* contains not merely a record of several cases of it, but also the first attempt at a physiological explanation of its cause. Especially interesting is one observation (No. lxxxvi.), under the heading "Local paralysis of the muscles of the extremities."† "These affections of particular muscles, or classes of muscles, imply a very partial disorder of the nerves. A disease of the brain, or a disease in the course of the nerve, must influence the whole limb, or that portion of it to which

* Bell, "Physiologische und pathologische Untersuchungen des Nervensystems;" übersetzt von Romberg, Berlin, 1832.

† *L.c.*, p. 364.

the nerve or nerves are distributed. But in these cases particular subdivisions of the nerves, included in the same sheath, or running the same course, are affected. I am inclined to attribute such partial defects to the influence of visceral irritation. In that case *it must still be the influence of the sympathetic nerve which produces it*; and, yet, on the other hand, it seems impossible to account for such entire loss of motion without the intermediate influence of the brain." Insufficient as Bell's argument must appear to us now, the first reference to the sympathetic nerve as the seat of the primary lesion, is not without interest. This allusion appears, nevertheless, to have met with but little attention, as Abercrombie describes the disease as a local nervous affection, and Romberg as a spinal paralysis, while the later authors regard it as of myopathic origin (Aran), or as an atrophy of the anterior roots of the spinal nerves (Cruveilhier). Besides these, various other theories have been propounded, tracing it principally to changes in the spinal marrow, especially in the anterior columns (Lockhart Clarke, Charcot, &c.); these views are more or less generally accepted, and we will not now further discuss them.

To Schneevogt* belongs the merit of first drawing attention to the accompanying affection of the sympathetic, which, in earlier examinations, appears to have escaped observation. In the case investigated by him the brain and spinal cord were normal as far downwards as the fourth cervical nerve, and considerably softened, and extensively infiltrated with finely granular fat and granulation cells, from the fifth cervical to the second dorsal nerve; the posterior roots of the cervical nerves were unchanged, the anterior strikingly thinned, especially the five upper, which consisted only of some very slender filaments. The cervical part of the sympathetic was converted almost into a cord of adipose tissue, in which the nerve-fibres were replaced by flat cells containing crystals. The cervical ganglia had almost completely degenerated into fatty tissue, while the thoracic part of the sympathetic likewise contained some fatty matter. The spinal ganglia and vagus were normal. Amongst the peripheral nerves the left ulnar especially showed striking changes. Schneevogt accordingly feels bound to support the doctrine that the disease is of a central origin, and includes both an

* "Niederl. Lancet," Sept. und Okt., 1854, p. 218. See Schmidt's "Jahrbücher," 1855; 87^{ter} Band, p. 179.

affection of the anterior roots and a disturbance of the sympathetic, or of the innervation of single ganglia.

Whilst in most of the later cases, with the sole exception of Schneevogt's, the examination of the sympathetic and ganglia seems to have been neglected, Jaccoud,* on the contrary, undertook this investigation in two cases, and attained positive results.

The two patients in question died in August and September, 1864, the one of asphyxia, from paralysis of the inspiratory apparatus, the other of gangrene of the lungs. In both there was atrophy of the anterior roots in all the cervical and the three or four upper dorsal nerves. On microscopical examination the white and grey substances of the spinal medulla were found unchanged. *On the other hand the sympathetic in both cases showed a fibro-fatty degeneration. The whole cervical part was transformed into fibrous connective tissue of decidedly old standing, in which were seen many spots of fatty infiltration; the nerve fibres were secondarily atrophied, and that to a greater degree than in the spinal roots. The ganglion cervicale supremum was only in the first stage of the process, there being considerable hyperplasia of the cortical and interstitial connective tissue, but no atrophy of the nerve elements, the fibres, as well as the ganglionic cells, appearing intact. In the rami communicantes was found atrophy of a nature corresponding to the condition of the anterior roots; the median nerve contained, amongst many healthy fibres, also some that were pathologically changed (simple atrophy, absence of the medullary sheath, and even of the axis cylinder). Jaccoud regards it as unquestionable, from the stages of the different processes, that the disease began in the cervical part of the sympathetic, and spread thence both centripetally (by the rami communicantes and the anterior roots) and centrifugally (as is indicated by the partial affection of the median nerve).*

Changes in the sympathetic are also mentioned by Swarzenski (atrophy of the trunk and of both upper ganglia), and by Duménil (fibro-fatty degeneration of the cervical and thoracic parts, considerable hyperplasia of the connective tissue, atrophy of the nerve-fibres, and regressive metamorphosis of the ganglionic cells).

In the greater number of recorded cases no accurate examination of the sympathetic system was made; its integrity, however, is specially affirmed by many good observers—

* Jaccoud, "Bulletin de la soc. méd. des hôpitaux de Paris;" Union méd. 1865 (T. xxv.), No. 4, p. 60. See also "Leçons de clinique médicale," p. 361.

Landry, Frommann, Menjaud, Hayem, Charcot and Joffroy, Duménil (in three cases), and Friedrich (in six cases).

Lubimoff,* on Charcot's suggestion, thoroughly examined the sympathetic—the cervical and thoracic parts, and abdominal ganglia—in two cases, one of protopathic progressive muscular atrophy, and one of a secondary nature, connected with lateral amyotrophic sclerosis. In the first case only the intervertebral ganglia exhibited appreciable changes, which, nevertheless, as the patient had at the same time suffered from tuberculosis, were probably due to this complication; the state of the sympathetic was a negative one. Also in the second case only some intestinal ganglia showed similar changes affecting the nerve and connective tissue cells, namely granular pigmentation of the latter.

Thus, to the five positive results are opposed at least sixteen completely, or almost completely, negative statements with respect to the sympathetic.

Let us now see if the clinical course of progressive muscular atrophy permits us to draw more positive conclusions regarding the eventual implication of the sympathetic than the few and contradictory items of information supplied by the autopsies hitherto made.

Remak† discovered that, in treating patients suffering from progressive muscular atrophy by galvanism, he could produce spasmodic movements in the atrophied muscles when the positive electrode was placed in an "irritable zone" reaching from the first to the fifth cervical vertebrae (especially in the fossa carotica, or in the triangle between the lower jaw and the external ear), and the negative electrode below the fifth cervical vertebra. Concerning these well-described, very remarkable phenomena, we will merely observe that the movement always took place on the side opposite to that on which the positive electrode was placed; when both were placed on the middle line the movements appeared on both sides; and when the current was very weak they occurred only in the most paralysed muscles. Remak regarded these spasmodic movements, which he afterwards named "Diplegic,"‡ as of reflex origin, produced through the ganglion cervicale supremum of the sympathetic, more especially as the entrance of the current was felt by the

* "Archiv. de Phys. normale et pathologique," I. Sér., 1874, p. 889.

† Remak, "Oesterr. Zeitschrift für practische Heilkunde," 1862, p. 1 and 29. See also "Application du courant constant au traitement des névroses," Paris, 1865, p. 26.

‡ Remak, "Application du courant constant," p. 28.

patient behind the eyeball. He holds that progressive muscular atrophy is a disease of the sympathetic ganglia, or also of the cervical part of the spinal cord. In that way may be explained the irregular progress of the muscular atrophy, inasmuch as in the centres the ganglionic cells, on which the nutrition of the muscles depends, have an arrangement different from that of the nerve-fibres in the peripheral trunks which are connected with them; further, this also explains the frequently simultaneous occurrence of "neuro-paralytic inflammation," which consists particularly in painful swelling of the joints (arthritis nodosa), and which yields to galvanic treatment of the sympathetic. Remak also includes the so-called reflex paralysis amongst the paralyzes of the sympathetic; probably also certain forms of hysterical, saturnine, and diphtheritic paralyzes.

We pass over Remak's other statements which refer more particularly to therapeutics, as we are now more especially treating of the pathogeny of the disease, and will again discuss, at the end of this sketch, its treatment by means of the so-called galvanization of the sympathetic.

Remak's observations regarding "diplegic movements" were confirmed by M. Meyer* and Drissen† in a case of progressive muscular atrophy. Fieber,‡ on the contrary, could not produce these movements in a similar case by following Remak's method. Benedikt§ also states that in the cases treated by him the diplegic movements were wanting, but, nevertheless, recommends galvanization of the sympathetic, in connection with other methods of galvanization—a plan of treatment the importance of which is fully proved by his valuable record|| of cases. Erb¶ also states that he has not been able to obtain the diplegic contractions in the manner described by Remak; it does not appear, however, that he experimented on actual cases of *progressive* muscular atrophy. Though in most of our cases of this disease the diplegic movements could not be excited, we were able, with little difficulty, to demonstrate them in *one* single case. With regard to the conditions which give rise to these movements, and their symptomatic explanation, we differ entirely from Remak. The relation, affirmed by Remak and adopted by

* M. Meyer, "Die Elektrizität in ihrer Anwendung auf practische Medicin." 3te Auflage," 1868, p. 219.

† Drissen, quoted in above book, p. 219.

‡ Fieber, "Berl. klin. Wochenschrift," 1866, No. 25, p. 261 (ix. Versuch).

§ Benedikt, "Elektrotherapie," ii. Abth. (Wien, 1866), p. 389.

|| "Ibidem," p. 389-412.

¶ Erb, "Arch. für klin. Medicin," 1867, iii. Band, p. 356.

Fieber and M. Meyer, of these motor phenomena to the sympathetic ganglia, appears to us, both on physiological and empirical grounds, to be in no way warranted. In none of the cases in which diplegic movements were observed by us (in progressive muscular atrophy, saturnine paralysis, and hysterical atrophy), did we find that they occurred specially or exclusively in the way stated by Remak, and we thus do not consider ourselves justified in adducing them as evidence of abnormality of function or condition in the sympathetic nerve.

More important in this relation is the occurrence of disturbances of the innervation of the eye, which can be traced to diseased action in the oculo-pupillary fibres distributed in the cervical sympathetic. In this category may be included some cases of progressive muscular atrophy recorded by Bærwinkel,* Voisin† (from Bouillaud's Clinique), and Menjeaud.‡

Voisin's case was that of a man aged 44, who had suffered from the disease seven or eight years, first in the left, and then in the right arm. For three or four weeks he felt slight spasmodic movements in the upper eyelid, frequently having the sensation as if a grain of sand were in the eye; during this time also his sight became weaker. The left pupil was only half as large as the right, both responded normally to the stimulus of light, and dilated on pinching various parts of the body. The left cornea was flattened so that its highest point lay 1mmtr. nearer the iris than on the right side. After some months the same changes appeared also in the right eye; both pupils were then equally small and sluggish in action, the cornea in both eyes equally flattened, and vision on both sides weak.

In Menjeaud's case,* in which the atrophy was confined chiefly to the region supplied by the median and ulnar nerves on both sides, there was considerable contraction of the left pupil. Post-mortem examination revealed the existence of atrophy of the anterior roots of the lowest cervical and uppermost dorsal nerves, especially on the left side. The sympathetic and its ganglia were normal.

In explanation of the phenomena occurring in the eye in his case, Voisin refers to Claude Bernard's experiments, in which division of the anterior roots of the two lowest cer-

* Bærwinkel, "Prager Vierteljahrschrift für pract Heilkunde," 1858, lix., p. 133.

† Voisin, "Gaz. hebdomadaire," 1863, No. 37; "Gaz. des hôp.," 1863, No. 110, p. 437.

‡ Menjeaud, "Gaz. des hôp.," 1866, No. 3, p. 10.

* L. c.

vical and the two uppermost dorsal nerves was followed by contraction of the pupil and flattening of the cornea. As in the case observed by him the muscles affected were principally those supplied by the median and ulnar nerves, and as these take their origin in part from the spinal nerves just named, he infers that the disturbance of nutrition proceeded centripetally from the peripheral nerve fibres to the anterior roots, and gave rise to secondary atrophy of the latter. The non-participation of the sympathetic in causing the oculo-pupillary phenomena in the above case is to be inferred, according to Voisin, from the circumstance that vaso-motor disturbance, especially increase of temperature in the affected parts, had not been noticed; he thus adopts Bernard's view, that the nerves controlling the vessels of the extremities have their origin entirely in the ganglia of the sympathetic and join the nerve trunks only outside the vertebral column.

From the occurrence of oculo-pupillary phenomena in progressive muscular atrophy, without simultaneous increase of temperature of the paralysed upper extremity, one cannot draw a just conclusion either regarding the participation of the anterior roots or the non-participation of the sympathetic, even if Voisin's record were on all points less inexact than it really is.

The occurrence of oculo-pupillary symptoms in the affection in question, however, is rare, if not quite exceptional. We find it mentioned only in a few instances by Bergmann, M. Rosenthal, and Friedreich. Duchenne states expressly, in discussing Voisin's case, that he had not once met with these phenomena. We also, in all our cases, which were a long time under observation, failed to discover disturbance of innervation in the eye.

It is only in Schneevogt's case, among all those in which degenerative changes in the cervical sympathetic were found, that contraction of the pupil is mentioned as a symptom. Here, as we have seen, there was not only fatty degeneration of the sympathetic, but atrophy of the anterior roots, involving specially the five upper cervical; there was also softening of the spinal cord from the fifth cervical to the second dorsal nerves—exactly in the region of the centrum ciliospinale inferius. The contraction might thus arise either from the diseased sympathetic or from the central medullary affection. Jaccoud's record of the cases in which he had a post-mortem examination, unfortunately contains nothing about the symptoms and course of the disease as they came under his notice only a few days before death.

We thus, in the meantime, get little aid, either from anatomical research or physiological analysis of symptoms, in our inquiry regarding the nature of progressive muscular atrophy. We can give, therefore, no definite opinion as to the part (though it is probably a not unimportant one) played in it by the sympathetic system. *Is the sympathetic usually affected at all? And if it is, is it through centripetal conduction of the primary muscular disease to the peripheral nerves, the spinal roots, and the rami communicantes? Or, contrariwise, is the sympathetic affection the primary one, spreading centrifugally to the peripheral nerve trunks and muscles, and centripetally to the spinal centres?* So run the questions which will have to be answered in the future. It is to be hoped that, should opportunity offer for making post-mortem examination, the investigation of the sympathetic may not be neglected, and that it may lead to more definite results; very specially should its condition be compared with that of the other central and peripheral nervous apparatuses, with particular reference to the nature and date of the disease; and, as is also indicated above, greater attention must be paid to the occurrence of oculo-pupillary and vasomotor phenomena.

With reference to therapeutics, we will, in conclusion, mention that in one case of severe progressive muscular atrophy reported by Nesemann,* galvanization produced a temporary improvement; but later a relapse took place, which did not yield to the same treatment. As by Remak, so by Benedikt, M. Meyer, Guthzeit, Erb (according to Friedreich) and others, favourable results have been obtained from galvanization of the sympathetic. M. Rosenthal and we ourselves have never seen much good follow from this treatment.

Muscular Hypertrophy.—This disease is known under various names, such as "*Muscular hypertrophy*," "*Lipomatosis musculorum luxurians progressiva*" (Heller); "*Atrophia musculorum lipomatosa*" (Seidel); "*Paralysie musculaire pseudo-hypertrophique*," or "*Paralysie myosclérosique*" (Duchenne).

Like progressive muscular atrophy, which is often combined with it, it has sometimes been regarded as a primary myopathic affection, and at other times as a neuropathic disease. The first-mentioned opinion was held by Spielman,† whilst Duchenne,‡ and after him Stoffella,§ without very obvious

* Nesemann, "Berl. klin. Wochenschrift," 1868, No. 37.

† Spielmann, "Gaz. méd. de Strasbourg," 1862. Mai, No. 5, p. 85 ff.

‡ Duchenne, "Electrisation localisée," 2 edit., 1861, p. 334.

§ Stoffella, "Zeitschrift der Gesellschaft der Aertzte," in Wein, 1865, Heft. 1, p. 85 ff.

reasons, argued that it was of cerebral origin. Griesinger* assumed that it was a disease of the vasomotor nerves, and Benedikt† mentions it among the "*trophoneuroses*," and believes one of his cases to have been of the nature of a *paralysis of the sympathetic*. In this case the hypertrophy affected principally the muscles of the right shoulder (the deltoid, pectoralis major, teres major and minor, serratus anticus major); moreover, *the right side of the face was redder, and perspired more freely than the left, the pupil was dilated, and the sympathetic tender to pressure*. Besides Menjon's‡ case, which was brought under this category by Seidel, we have only three detailed accounts of post-mortem examinations in this disease. The first is one recorded by Eulenburg,§ in which Cohnheim made the examination. The result, with respect to the nervous system was perfectly negative; microscopic examination revealed no pathological changes in the brain, spinal medulla, peripheral nerves, or sympathetic system. Barth,|| on the other hand, found changes in the spinal cord and peripheral nerves which he believes are only secondary. Unfortunately no examination of the sympathetic was made. In a case investigated by Charcot, the state of the spinal cord and peripheral nerves was entirely a negative one. Therapeutically it may only be mentioned that Benedikt thinks he obtained considerable improvement in three cases by galvanization of the sympathetic. In one of the cases already quoted, not only the symptoms in the head, but also the hypertrophy disappeared, so that the patient could resume work. Chvostek¶ states, however, that the prolonged use of electricity is followed, at most, merely by arrest of the morbid process. We ourselves got no successful result from the galvanic method of treatment in one case; it was, however, very severe and of old standing. But in two cases (in two young girls), which were some time under our observation, faradaic electrization of the atrophied and of the pseudo-atrophied muscles was followed by improvement, which was not apparent on galvanizing the sympathetic. O. Berger,** who also considers the disease a trophoneurose, faradised the

* Griesinger, "Archiv. der Heilkunde," 1864 (6ter Jahrgang), p. 171.

† Benedikt, "Elektrotherapie," Wien, 1869, p. 186 ff.

‡ Menjon, "Med. Chir. Transactions," Vol. liii., 1852, p. 73 ff. See also Seidel, "Die Atrophia musculorum lipomatosa," Jena, 1867, p. 64.

§ Eulenburg, "Berl. klin. Wochenschrift," 1865. No. 50; *ibidem* 1866. No. 37.

|| Barth, "Archiv. der Heilkunde," 1871, Bd. xii., p. 121.

¶ Chvostek, "Oesterr. Zeitschrift für practische Heilkunde," 1871. No 38-40.

** Berger, "Deutsches Archiv. für klin. Medicin," 1872. Band ix., p. 363.

hypertrophied muscles and galvanised the cervical sympathetic for weeks in two cases without making any impression on the disease. Erb records the same experience.

VII.—EXOPHTHALMIC GOÏTRE.—(BASEDOW'S DISEASE.)
(GRAVES' DISEASE.)

This name is given to a group of symptoms, discovered by Parry in 1825, but first accurately described by Basedow* in 1840, including palpitation of the heart, swelling of the thyroid gland, and protrusion of the eyeball (exophthalmos). These three cardinal symptoms are usually found together, but any one of them may be wanting. The order in which they are usually developed is—first, palpitation of the heart (with or without atrophy), then goitre, and finally exophthalmos; it seldom occurs that all the symptoms appear suddenly and simultaneously, or that the tumefaction of the thyroid body is noticed before the heart affection. Other disturbances, especially in the nervous system and the female generative organs, are occasionally observed, but they are merely of secondary importance.

Concerning the nature and origin of this disease, many different theories have been promulgated. It was at first most naturally regarded as a special form of chlorosis or anæmia; but this idea must be given up, as it is often met with in men and in children, and in women beyond the climacteric period, and frequently in those whose catamenia are quite regular. The palpitation of the heart has been thought to be the cause of the other symptoms—a theory that is negatived by the simple fact that the accelerated and more powerful action of the heart in cases of lesion of the cardiac valves never leads to goitre and exophthalmos. The seat of the disease has further been placed in the nervous system, especially in the spinal cord. When Claude Bernard made known the phenomena produced by division of the cervical sympathetic in animals, it was noticed that there were certain resemblances between them and the symptoms of exophthalmic goitre, and the inference was drawn that the disease was caused by functional disturbance in the cervical sympathetic (Koeben,† Aran,‡ Trousseau,§ &c.) Since that time this theory has found wider acceptance for the reason that, in a

* Casper's "Wochenschrift," 1840. No. 13 and 14.

† "De exophthalmo a struma cum cordis affectione." Inaugural Dissert., Berlin, 1855.

‡ "Gaz. hebdom.," 1860. No. 49.

§ "Gaz. Med.," 1862, p. 474.

small number of cases of Graves' disease anatomical changes have actually been found in the cervical sympathetic. Before we relate these cases individually, we will try to show in what manner the principal symptoms of this disease may be compared with the phenomena occurring after division of the cervical sympathetic in animals. Division of the cervical sympathetic produces dilatation of the vessels, and, in consequence of there being more blood in the parts, a considerable elevation of temperature on the corresponding side of the head. Analogies for both of these experimental facts are found in Basedow's disease. The dilatation of the vessels indicates its presence in the strongly pulsating, frequently tortuous and prominent little arteries in the region supplied by the carotid, but especially in the swollen thyroid gland. The sudden occurrence of the goitre in the course of a few days, the softness of the swelling, the perceptible pulsation of the thyroid arteries, the loud blowing sounds heard over the same, the rapid increase and decrease of the tumour, according to the force of the contractions of the heart, and the engorged veins frequently seen on the surface of the gland—all these facts point to the conclusion that we have to do chiefly with a dilatation of the vessels distributed in the thyroid body, a conclusion fully borne out by anatomical investigation.*

Although we find, in the enlargement of the thyroid vessels, a resemblance to the dilatation of the vessels after division of the cervical sympathetic, yet this similarity is not evidence of a complete analogy; for then proof would be required that division of the sympathetic also leads to swelling of the thyroid gland. That animals, as well as men, may be the subjects of swelling of this gland is well known. In districts in which goitre occurs endemically amongst the people, Baillarger† has observed it amongst the animals, especially in mules, seldomer in horses and dogs. Boddaert‡ has also produced a swelling of the thyroid body in rabbits and guinea-pigs by ligaturing the internal and external jugular and inferior thyroid veins. Regarding the second of the constant phenomena appearing after division of the sympathetic in the neck—the elevation of temperature—something similar has been found in exophthalmic goitre, since special attention

* Naumann, "Deutsche Klinik," 1853. No. 24. F. Banks, "Dublin Hosp. Gaz.," 1855. No. 9. Fournier et Ollivier, "Union Méd.," 1868, p. 95.

† "Du goitre exophthalmique chez les animaux domestiques." Comptes rendus, 1862. Tome lv., p. 475. See also Virchow, "Die krankhaften Geschwülste," 1867, Bd. iii., p. 57.

‡ "Extrait du Bulletin de la Soc. de Méd. de Gand.," 1872.

has been directed to the subject; thus, Paul* has found an increase of $\frac{1}{2}$ -1° C., Teissier† of 1-2° C.; and Cheadle‡ states, in a record of eight cases, that he has always observed a rise in the temperature. We ourselves, in nearly all our cases, have noticed an increase (at least temporarily) of $\frac{1}{2}$ -1° C.; and in one case, that of a girl 20 years of age, who was under treatment about nine months, the temperature in the axilla always amounted to 38·2-38·8° C. Nevertheless there are cases in which the temperature is normal, as recorded by Charcot§ and Dumont.|| It is evident that this elevation of temperature is of comparative value as a proof of pathological analogy to that occurring after division of the cervical sympathetic only when observed in an uncomplicated case of Basedow's disease, and not caused by any concomitant febrile affection. Those suffering from this disease feel the rise in temperature subjectively as heat, even when objectively it is very slight; this is often accompanied by increased secretion of perspiration.

With reference to the cause of this increased temperature, nothing is opposed to the idea that it is to be sought in the increased blood-supply resulting from dilatation of the vessels; it is not, however, locally confined, like that produced experimentally by dividing the sympathetic, but is equal in both axillæ and in both auditory meatuses. The sympathetic is clearly involved on both sides, as is shown by the facts that the vessels on both sides are equally filled, and that the exophthalmos is bilateral.

The second principal symptom in exophthalmic goitre which we have to compare with the results of division of the sympathetic is exophthalmos. It is seldom unilateral, but usually appears in both eyes at once, though not always with equal intensity.¶

After division of the cervical sympathetic no exophthalmos occurs, but rather a sinking of the eyeball within the orbit. But if the central end of the divided nerve be subjected to electrical stimulation the eyeball is protruded, and we have

* "Berliner klinische Wochenschrift," 1865. No. 27.

† See Trousseau, "Clinique méd.," T. ii., p. 540.

‡ "Lancet," 1869. No. 25.

§ "Gaz. méd.," 1856, p. 600.

|| "De Morbo Basedowii." Inaug. Dissert., Berlin, 1863, p. 27.

¶ Such cases have been observed by Mackenzie, quoted by Fischer, "Archives générales de Méd.," 1859, p. 652; by Förster, quoted by Lebert, "die Krankheiten der Schilddrüse und ihre Behandlung," Breslau, 1862, p. 309; by Schnitzler, "Wiener, Medicinal-Halle," 1864, No. 27; by Chisholm, "Med. Times and Gaz.," 1871, No. 1; by Emmert, "Archiv. für Ophthalmologie," 1871, Bd. xvii., p. 218.

genuine exophthalmos. It has already been explained in the physiological part of this work, that this proptosis is due to the action of Müller's unstriated ocular muscles and of some other unstriated muscles discovered in the upper and lower lids and in the orbital aponeurosis, all these being supplied by the sympathetic nerve, and thrown into contraction on irritating it. The occurrence of exophthalmos in this disease might be explained, if we were to assume the existence of a condition of irritation in the oculo-pupillary fibres of the cervical sympathetic. Thus, as we had to assume a state of paralysis of the vasomotor fibres of the cervical sympathetic (analogous to division in animals) to explain the dilatation of the vessels in the thyroid gland, and the rise in temperature, we have to do with two opposite conditions—paralysis of the vasomotor and irritation of the oculo-pupillary fibres of the cervical sympathetic. This assumption contains nothing arbitrary. We know, as we formerly explained, that the vasomotor and oculo-pupillary nerves have entirely different centres, and that we can experimentally educe independently of each other the appearances depending on disturbance of one or other class of fibres. If we now suppose that Graves' disease rises from an affection of the nerve centres, we may well conceive that the centre for the oculo-pupillary fibres of the cervical sympathetic (*centrum cilio-spinale*) is in a condition of irritation while, on the contrary, the centre for the vasomotor fibres is in a state of paralysis. Even though we regarded the cause of exophthalmic goitre as not central, but peripheral, and situated in the cervical sympathetic, there is nothing forced in the above supposition, that the oculo-pupillary fibres are in a condition of irritation, and the vasomotor fibres in a state of paralysis. We find many analogies in the pathology of the peripheral nervous system. Thus, in neuritis a state of irritation in the motor part of a nerve (spasm) may co-exist with one of paralysis in the sensory portion (anæsthesia); or, on the other hand, we may have paralysis of the motor and hyperæsthesia of the sensory filaments. Even in the same fibres we may often find opposite conditions; in sensory fibres for example, both diminished and increased sensibility (anæsthesia dolorosa), and in motor fibres diminished and increased motor power (paresis, or paralysis associated with slight spasmodic movements).

We have endeavoured to show the relation existing between Basedow's exophthalmos and the cervical sympathetic, inasmuch as we pointed out that in animals exoph-

thalmos follows irritation of that part of the sympathetic system. The question now rises whether we are justified in regarding this exophthalmos as due *entirely* to a state of irritation in the sympathetic, and thus genetically the same as the exophthalmos produced experimentally. Such an assumption we cannot make. Permanent exophthalmos, such as that of Basedow's disease, is only possible when the unstriped ocular muscles, which have their innervation from the sympathetic, are in a state of persistent tetanic contraction, that is, when there is a permanent state of irritation in the nerve fibres supplying these muscles. There is, however, no physiological analogy to warrant the adoption of such an opinion. Every state of irritation in a nerve passes gradually into the opposite condition, that of paralysis. We cannot, therefore, unconditionally claim the exophthalmos occurring in animals on irritating the cervical sympathetic by an electric current as an explanation of Basedow's exophthalmos. It is also not easy to conceive that the effect of spasm of these muscles, so slightly developed in man, should be to produce such an extreme degree of proptosis as is often met with in exophthalmic goitre; for even with the strongest electrical stimulation of the cervical sympathetic, a degree of irritation which can never occur under physiological conditions, there is never produced such marked exophthalmos as we find in this affection. Thus we may fall back on the view of the earlier authors, that the *congestion* (venous hyperæmia) and *development of fat* in the cellular tissue of the orbit tend to aid in causing protrusion of the eyeball. That there is congestion in the orbit during life is indicated by the broad and tortuous retinal veins,* and by the facts that when the palpitation of the heart abates the exophthalmos becomes less marked, that on increase of the heart's action the eyeball becomes again more prominent, and further, that the eyeball sinks into the orbit on slight pressure with the finger and after death. There are also various reasons for supposing that the eyeball is pressed forward by a cause of a mechanical nature. In persons who have died from strangulation there is exophthalmos in consequence of venous engorgement, and the same is produced in animals by ligation of the jugular vein. Further, new-born children whose birth has been accomplished only after prolonged labour, or by aid of instruments, present a slight degree of exophthalmos, the result

* v. Gräfe, "Archiv. für Ophthalmologie," 1857, p. 292.

of the pressure on the head, which prevents the free return of blood. The same is observed in women in severe labour—a fact which was well known to the ancients. Exophthalmos may arise from various other mechanical causes which lead to serous infiltration of the retrobulbar connective tissue (as in dropsy), and from congestions in the head. Many conditions (not including tumours of the brain), such as violent and prolonged bodily exertion, and convulsions, may also give rise to exophthalmos by increasing the pressure in the veins. Demarquay* has collected several such cases from general medical literature. As regards the abnormal development of fat as a factor tending to produce exophthalmos, it has often been demonstrated† in at least double the normal quantity, and sometimes even more. Thus, it is extremely probable that all three conditions—spasm of the unstriated orbital muscles, venous hyperæmia, and increase of the fatty tissue in the orbit—co-operate in the production of exophthalmos.

We have still to consider a peculiar phenomenon to which v. Gräfe‡ was the first to direct attention, and which unquestionably indicates the participation of the sympathetic in the production of the disease. In health when the plane of vision is altered, when the eye is turned upwards or downwards, the upper eyelid follows closely the movements of the eyeball; in exophthalmic goitre this consentaneous movement of the eyelid is wanting. According to v. Gräfe it is not caused by the prominence of the eye, as the movements of the lid remain intact in exophthalmos from other causes (from tumours of the orbit, for instance); on the other hand, this mobility is lost even in the slightest degrees of the exophthalmos of Basedow's disease. This symptom, too, may disappear in the course of the disease, both spontaneously and on using narcotic injections without any improvement in the proptosis. Gräfe regards it as of great importance in the recognition of the slighter forms of the affection, those cases in which the prominence of the eye does not exceed physiological bounds and in which the goitre is wanting. The cause of this deficient mobility of the

* "Traité des Tumeurs de l'orbite." Paris, 1860, p. 157-223.

† Basedow, "Casper's Wochenschrift," 1848, p. 775; Heusinger, "Casper's Wochenschrift," 1851, p. 52; Brück, "Deutsche Klinik," 1862, p. 207; Naumann, "Deutsche Klinik," 1853, p. 269; Laqueur, "De Morbo Basedowii," Inaug. Diss., Berlin, 1860, p. 12; v. Recklinghausen, "Deutsche Klinik," 1863, p. 288; Peter, "Gaz. hebdom," 1864, p. 181; Fournier et Ollivier, "Union méd.," 1868, p. 95.

‡ "Deutsche Klinik," 1864, p. 158; and "Berliner klinische Wochenschrift," 1867, No. 31.

eyelid is, according to him, to be sought for in disturbed innervation (spasmodic contraction) of Müller's unstriated orbital muscles.

Another symptom in exophthalmic goitre which tends to connect it with the sympathetic is the occurrence of various inflammatory and ulcerative affections of the eye, which fortunately appear but seldom, principally among men,* but also occasionally amongst women.†

v. Gräfe considers the above-mentioned insufficiency of the eyelid as the chief cause of these inflammatory affections: on account of the deficient mobility of the upper eyelid when looking down, as in reading, a part of the cornea remains uncovered, the conjunctival sac becomes dry and the veins dilated, and thus inflammation and even chemosis are produced. But this affection of the lids cannot be regarded as the sole cause of the dry state of the eyeball and the consequent inflammatory symptoms, since the same circumstances in paralytic lagophthalmos (as in facial paralysis) generally leave the eye unaffected. v. Gräfe thus holds that Basedow's ophthalmia is principally of a neuromyopathic character, caused by disordered function of the sympathetic fibres of the trigeminus. This notion receives confirmation by the observation that in severe cases of Basedow's disease the sensibility of the cornea is lowered.

In comparing the symptoms of Basedow's exophthalmos with the phenomena following division of the cervical sympathetic, we have still to advert to the pupil. Dilatation of the pupil accompanies every case of exophthalmos experimentally produced; in that of exophthalmic goitre, on the contrary, it is entirely absent. v. Gräfe has recorded its absence in about two hundred cases. When, opposed to this large number, some observers‡ state that they *have* seen dilatation

* Basedow, "Casper's Wochenschrift," 1840, p. 222; Praël, "Archiv. für Ophthalmologie," 1857, Bd. iii., p. 201; Naumann, *loco citato*, p. 286; v. Gräfe, "Archiv. für Ophthalmologie," 1857, p. 285; and "Berliner klin. Wochenschrift," 1867, No. 31.

† Lavrence, "Gaz. des hôp." 1858, p. 198; Tatum, "Med. Times and Gaz.," 1864, 23 January, p. 39; Teissier, "Canstatt's Jahresbericht," 1864, Bd. iv., p. 173; Patchett, "Lancet," 1872, June 15.

‡ Romberg and Henoch, "Klinische Wahrnehmungen und Beobachtungen," Berlin, 1851, p. 182; Reith, "Med. Times and Gaz.," 1865, p. 521; Friedrich, "Lehrbuch der Herzkrankheiten," Erlangen, 1867, p. 312; Troussneau, "Clinique méd.," Paris, 1868, Tome ii., p. 536; Fournier et Ollivier, "Union méd.," 1869, p. 93; Gildemeester, "Archiv. für die holländischen Beiträge zur Natur und Heilkunde," Utrecht, 1864, Bd. III., p. 416 and 420; Cheadle, "Lancet," 1869, No. 25; Emmert, "Archiv. für Ophthalmologie," 1871, Bd. xvii., p. 203.

of the pupil in a few cases, this symptom can scarcely be considered as having any pathological connection with Basedow's disease; most likely these patients were myopic. From the absence of dilatation or other abnormality of the pupil, we conclude that the pupillary fibres of the sympathetic are not involved in this affection.

The third principal symptom in exophthalmic goitre is the increased action of the heart, which may certainly be regarded as depending on functional disturbances in the cervical sympathetic. That nerve contains fibres whose function is to accelerate the heart's action, and electrical irritation of these increases the number of the heart's contractions. For further details on this subject we refer to the physiological part of this essay. The increased action of the heart in Graves' disease may be explained by assuming the existence of irritation of the cervical sympathetic; but, as we have already pointed out, while discussing exophthalmos, the assumption of such a *persistent* state of irritation in order to explain the permanency of the heart affection is physiologically inadmissible, as this condition must soon pass into that of paralysis. The increased action of the heart, however, may be interpreted as due to a variety of *paralysis* in the cervical sympathetic; paralysis of the cardiac sympathetic nerve fibres leads to dilatation of the cardiac vessels (the coronary arteries), and therefore to a greater flow of blood to the muscular tissue of the heart, and thus to stimulation of the cardiac ganglia.

Having gone over the principal symptoms of Basedow's disease, and shown their relation to the phenomena observed in certain experiments performed on the cervical sympathetic, we come now to the description of the pathological anatomical changes which have been found in the sympathetic after this disease. The number of facts is certainly small; but it must be remembered that the opportunities for making post mortem examination have been comparatively few, and that it is only lately that attention has been directed to the sympathetic system as connected with the affection. The cases which have come to our knowledge are the following:—

1.—A case from Trousseau's Clinique, described by Peter.* It was that of a woman who, seven years before coming under observation in the Clinique, had received a violent shock by the sudden death of her father, this being followed, in one

* "Notes, pour servir à l'histoire du goître exophthalmique," Gaz. hebdom., 1864, No. 12, p. 180.

night, by exophthalmos, goitre, and palpitation of the heart. Five months later she died comatose, after an apoplectic seizure. At the examination the upper and middle cervical ganglia were found normal, but the lower, especially on the right side, considerably enlarged and injected. Microscopic examination showed a marked development of connective tissue in the lower cervical ganglion, whilst the nerve tissue (ganglionic cells, and nerve filaments) had become considerably atrophied.

2.—A case described by Archibald Reith.* A man, 24 years of age, who had suffered some time from Graves' disease, died two days after being taken into hospital. The autopsy, performed by Dr. Beveridge, 22 hours after death, showed principally, as regards the cervical sympathetic a hypertrophy of the middle and lower ganglia; they were hard and firm, and were seen, under the microscope, to be infiltrated by greyish matter. The trunk of the sympathetic and the branches of the inferior thyroid and vertebral arteries were increased in size and tuberculous.

3.—In a case examined by Cruise and M'Donnel, and recorded by Moore,† the lower cervical ganglion was almost obliterated, and replaced by cellular and fatty tissue.

4.—In the case of a girl treated by Traube‡ during life, and examined by v. Recklinghausen, there was *remarkable thinning of the sympathetic and its ganglia*.

5.—Biermer§ found, in the case of a man, considerable atrophy of the sympathetic on both sides of the neck, especially the right.

6.—Virchow|| mentions having observed an increase of size, and interstitial thickening, of the cervical sympathetic, especially in the upper and lower ganglia.

7.—In the case of a scholar, 48 years of age, who had died in Würzburg of Basedow's disease, Geigel¶ states that both cervical sympathetics were surrounded by a thick sheath of fatty and connective tissue; the microscope, however, showed no change either in the nerves themselves or in the ganglia,

* Reith, "Exophthalmos—Enlargement of Thyroid Gland—Affection of Cervical Sympathetic," "Med. Times and Gaz.," 11 Novr., 1865, p. 521.

† William Moore, "Some remarks on the Nature and Treatment of Pulsating Thyroid Gland, with Exophthalmos," "Dubl. Quar. Jour. of Med. Science," 1865, p. 344-352.

‡ Traube and v. Recklinghausen, "Deutsche Klinik," 1863, No. 29, p. 286.

§ This case was communicated to us by letter.

|| Virchow, "Die krankhaften Geschwülste," Bd. iii., p. 81.

¶ Geigel, "Würzburger med. Zeitschrift," 1866, Bd. vii., p. 84.

except intense brown pigmentation of the latter; there was also no increase of the interstitial connective tissue.

8.—Knight* found, in examining the body of a man of 33 years of age, who had died of Graves' disease, that *the left lower cervical ganglion of the sympathetic was larger than the right, the connective tissue increased in quantity, while the nerve-cells appeared much smaller and less pigmented. In the middle and lower left cervical ganglia the nerve-cells were smaller than on the right side, while the nerve filaments in the left sympathetic were only half the size of those in the right.*

9.—Ganghofner† relates the case of a servant, 43 years of age, who was under his treatment for exophthalmic goitre, and whose body, after death, was examined by Klebs. *The left sympathetic in the neck appeared extremely atrophied in its lower part, measuring scarcely $\frac{1}{2}$ mm. in thickness; in the upper part it was thicker, about $7\frac{1}{2}$ mm.; above the middle ganglion it was normal in point of size, but unusually red. The atrophied spot was about 2 centimeters long. Below this was found the lower ganglion, about the size of a pea, from which emerged two very thin nerves, one passing forwards, the other backwards. Under the jaw and on the sympathetic lay a lymphatic gland, swollen and much reddened. The upper ganglion was normal.—On the right side the upper cervical ganglion was normal, the middle ganglion somewhat broader; below this the sympathetic became very thin, and passed into a small ganglion, from which issued some exceedingly slender filaments which followed the course of the vessels. Microscopic examination of the atrophied part of the sympathetic revealed the presence of atrophy of the nerve elements.* This case, so valuable because so carefully examined, is, as regards the condition of the sympathetic, analogous to those of Traube and Biermer, and partly also to Knight's, inasmuch as here also the prominent change is atrophy of the sympathetic.

Opposed to these positive statements are four cases in which no change was found in the sympathetic.

1.—In Paul's‡ case there was nothing abnormal in the thoracic and cervical parts of the sympathetic, nor in both lower cervical ganglia. Microscopic examination of longitudinal and transverse sections of the right lower ganglion, both fresh and after carmination, showed that the nerve

* Knight, "Boston Med. and Surg. Journal," 1868, 19 April.

† Ganghofner, "Prager Vierteljahrschrift," 1876, Bd. cxxx.

‡ Paul, "Berliner klin. Wochenschrift," 1856, No. 27.

filaments and ganglionic cells were of normal dimensions, with clearly marked nuclei and nucleoli, partly colourless, and partly pigmented.

2.—In a case recorded by Fournier and Ollivier,* and most carefully dissected by Ranvier, no alteration was found in the sympathetic, either on examination with the microscope or by the naked eye. The case is specially remarkable, as death occurred by gangrene of the extremities; no cause for the gangrene was found.

3.—Rabéjac† has described a case of exophthalmic goitre in a woman 37 years of age, which also ended fatally through gangrene of the extremities. Bouvier examined the sympathetic microscopically, and could discover nothing abnormal.

4.—We have to quote only one other case, that of Wilks;‡ in it the ganglia of the sympathetic showed no change, except that they were strikingly white in colour; microscopically there was nothing abnormal, except some increase in the quantity of the connective tissue fibres.

These negative results prove nothing against the assumption that Basedow's disease may be connected with functional disturbances in the sympathetic, because these may exist without being necessarily accompanied by any anatomically demonstrable change.

With respect to treatment, we mention only the effects of galvanization of the sympathetic. In 1867 we performed our first experiments on this subject, and found that, on using a very weak ascending current of only 6-8 elements, the frequency of the pulse fell from 120 to 90 per minute.

More lately Chvostek,§ M. Meyer,|| and others, got good results by galvanizing the sympathetic, especially as regards the exophthalmos and the goitre, whilst the influence on the heart was but slight and transitory. The improvement in the goitre and the exophthalmos, which was permanent, was also accompanied by improved general health, showing itself in abatement of the chlorotic symptoms, and reappearance of normal menstruation.

* Fournier et Ollivier, "Union méd.," 1868, No. 8 and 9.

† Rabéjac, "Du goitre exophthalmique," Thèse, Paris, 1869.

‡ Wilks, "Guy's Hosp. Reports," 1870, Bd. xv., p. 17 ff.

§ Chvostek, "Wiener med. Presse," 1869, No. 19; 1871, No. 41; 1872, No. 23.

|| M. Meyer, "Berliner klin. Wochenschrift," 1872, No. 39.

VIII.—ANGINA PECTORIS.

The disease described by Heberden (1768) as *Angina Pectoris*, which is sometimes also termed *Stenocardia*, is not an anatomically definable cardiac affection, but rather a group of symptoms, originating in very different and for the most part unknown conditions. Although, therefore, we include a short account of it in this essay, we do not wish to indicate it as our belief that it is caused exclusively by changes in the sympathetic system, but that it seems to us that the latter, inasmuch as it takes the principal part in the formation of the cardiac plexus—which is almost certainly the starting point of *angina pectoris*—is undoubtedly in some way connected with the occurrence of the phenomena under consideration.

Angina pectoris shows a marked tendency to occur in paroxysms, occasionally spoken of as *stenocardiac attacks*. It appears as a complication in the most diverse diseases of the organs of circulation, relatively most often in affections of the aorta (insufficiency of the aortic valves, atheroma, &c.). Parry, however, held that its most common cause is to be found in the changes (ossification and contraction) which take place in the coronary arteries as the result of aortic insufficiency. But *angina pectoris* is not unfrequently met with also in cases in which no alteration in the coronary arteries, or any other affection of the heart, is noticeable; and, on the other hand, ossification of the walls of these vessels has been found on examining the bodies of old people who, during life, had never suffered from anything resembling *stenocardiac attacks*. Thus, besides the organic form of *angina pectoris*, that which is associated with structural cardiac disease, some have recognised a nervous variety, designating it *hyperæsthesia (neuralgia) of the cardiac nerves*, especially of the cardiac plexus. Other authors affirm that the essential elements of the disease are excitement of the heart's action and the neuralgic character of the seizures. We adopt in its entirety the theory that *angina pectoris* is not simply a disorder of sensibility, but a complex sensoromotor neurose of the cardiac nerves. We consider it impossible, however, to particularise with certainty what nerves are concerned in the attack itself; the cardiac nerves anas-

tomose so freely, and spring from such widely separated parts of the nervous system, that the existence of an anatomical affection or functional disturbance confined to those branches of the cardiac plexus coming from the vagus or sympathetic nerve seems scarcely within the limits of possibility. No anatomical changes have yet been observed, associated with angina pectoris, in those parts where the nerves of the heart run separately—in the cardiac twigs of the vagus, for instance, or in the branches which emerge from the cervical ganglia of the sympathetic. The phenomena also of the attack itself, especially the disturbance of the heart's action, are so variable that we cannot ascribe them to the influence of any single system of cardiac nerves. If, for example, in one case we account for the acceleration of the action of the heart by assuming a state of irritation of the sympathetic nerve, this explanation will not meet those cases in which, during the paroxysm, the heart contracts more slowly than usual, or scarcely departs from its normal rate. In discussing angina pectoris, therefore, we cannot argue from the state of any *one* system of cardiac nerves, but can only analyse the symptoms physiologically, and endeavour to show, on experimental grounds, in what way the different systems of nerves *may* be concerned in producing the disease.

Here we meet with the difficulty that the symptoms of the stenocardiac attack have been described very differently by different observers. The neuralgic symptoms alone—pain of a paroxysmal character associated with great oppression and anxiety—are constant and pathognomic. The pain appears to rise in the neighbourhood of the præcordia, and to pass sometimes over the left side of the chest, sometimes along the sternum towards the *left* arm, more rarely into both arms.

The motor phenomena—those connected with excitement of the motor nerves of the heart—are much less characteristic of the disease than the above-mentioned sensory disturbances. Some have described the heart's action as nearly normal, some as increased in force and associated with palpitation and a full pulse, and others as diminished in force and accompanied by a small quick pulse. The last observation has given rise to the theory that angina pectoris consists of spasm of the heart, whereby the contractions are rendered very weak and incomplete; hence the term “steno-

cardia." Occasionally, however, while the heart *appears* to be acting vigorously, its real force is but slight, since, in spite of its apparently powerful contraction, the radial arteries show only a small degree of elevation and tension—at least, towards the end of the attack (Lauder Brunton *).

With respect, also, to the respiration, the observations on record are quite at variance with each other. It is sometimes stated to be quick, sometimes slow, and occasionally not at all involved. Our own experience leads us to adopt Parry's † conclusion, that the changes in the respiration are principally, perhaps even solely, due to the pain.

These contradictory statements may be explained by Eichwald's ‡ clinical observation, that even in one and the same patient the state of the respiratory and circulatory organs varies greatly, according to the duration of the attack and the exact stage at which the examination is made. Thus, in the same case the action of the heart may be found either excited and powerful or weakened, or there may be great dyspnoea or perfectly tranquil respiration. The more violent the attack the more marked may be this contrast. During the paroxysm, when the pain is most severe, the force of the heart's contractions seems to be lessened, while in the painless intervals it appears to be increased. Severe cases are marked by a succession of attacks, the patient, during the intermissions, being free of pain. Less acute cases consist sometimes of only *one* such paroxysm, in which the movements of the heart and respiratory organs may be nearly normal. In the physiological analysis of the phenomena, however, we must keep before our minds only the most typical cases, and in these the action of the heart is always more or less affected.

We thus regard the substernal pain, the feeling of anxiety, and the disturbance of the heart's action, as the essential symptoms of angina pectoris, and the changes in the respiration, for the most part, as only the result of the pain. How far these conditions may be ascribed to disordered innervation of the heart we will now attempt to show.

The *pain* which ushers in an attack has its origin, doubtless, in the cardiac nerve plexuses. This assumption is supported by the circumstance that the pain always rises in and

* "Lancet," 1867, July 27, p. 97.

† See Stokes, "Lehrbuch der Herzkrankheiten," p. 398.

‡ "Würzburger med. Zeitschrift," 1863, Band iv., p. 49. 2

remains fixed at the part corresponding to the situation of the heart, and here, also, is most intense. There is also no affection of any part in the immediate neighbourhood of the heart which gives rise to such striking and unmistakeable symptoms; those, especially, of gastrodynia (cardialgia) bear no resemblance to the paroxysms of angina pectoris, and may be excluded from further consideration. In its normal state the heart has certainly little sensibility; that, nevertheless, an irritation of its sensory nerves under pathological conditions should produce so much pain is as little to be wondered at as the like result occurring in other organs having their innervation from the sympathetic—in the stomach, intestines, &c., for example. The heart receives its sensory nerves, not only from the vagus, but also from the sympathetic; for when both vagi are divided, and the heart is subjected to mechanical irritation, the animals so operated on (rabbits), as Goltz * has noticed, give indications that they feel pain.

It thus appears pretty certain that the sympathetic, which forms such a large part of the cardiac plexuses, contains nerves of sensation. It has also been justly remarked that the nature of the pain in angina pectoris shows great analogy to that of irritation of the sympathetic nerves, as in affections of the gall-ducts (gall-stone colic) of the stomach (cardialgia), &c.

This neuralgia of the nerves of the heart is in some cases idiopathic, but is more often the result of direct irritation. One naturally thinks of such a theory in certain organic diseases of the heart, such as ossification of the coronary arteries and aortic valvular deficiencies; the cardiac plexus also lies so close to the arch of the aorta (below and behind it), with the aortic plexus very near it, that morbid changes going on in the walls of that vessel may readily cause direct mechanical irritation of the plexuses in the immediate vicinity. Why these acute attacks of pain occur only as paroxysms at certain intervals, whilst the hypothetical cause which gives rise to them continues uninterruptedly in operation, is as difficult to explain in this as in other paroxysmal neuralgias.

As regards the causes of the pain in those cases in which organic changes in the heart are wanting, we cannot even

* Virchow's "*Archiv*," 1863, 26th Band, p. 1.

guess what they are. It cannot have its *only* origin in the change in the heart's action, whether the power of the heart be increased or diminished, as the most extreme departure from the normal standard in that respect, both the increased action of hypertrophy and the decreased action in fatty degeneration, may indeed produce a sensation of anxiety, but is never associated with pain at all resembling that of angina pectoris. When the heart's action is affected to only a moderate degree it is very frequently not perceptible by the patient.

In explanation of the transmission of the pain from the præcordial region to the other parts of the body, especially to the left arm, we must devote a few words to the anatomical relations of the cardiac plexus, its composition, and its connections with the nerves of the arms and neck. The *cardiac plexus* is composed of the cardiac branches of the vagus and the cardiac nerves, the latter rising in the cervical ganglia and the first thoracic ganglion of the sympathetic. The *ganglion cervicale supremum* of the sympathetic, in which the *superior cardiac nerve* originates, has several connecting branches with the three or four upper cervical nerves, which, again, contribute to the formation of the cervical plexus; further, the superior cardiac nerve anastomoses in the neck with twigs from the descending branch of the hypoglossal nerve, which also receives filaments from the second and third cervical nerves. The superior cardiac nerve is thus seen to have intimate relations with a number of the cervical spinal nerves.

The *ganglion cervicale medium*, from which proceeds the *middle cardiac nerve*, communicates with the fifth and sixth cervical nerves, sometimes also with the vagus and phrenic nerves.

The *ganglion cervicale inferius*, from which the *inferior cardiac nerve* rises, sends branches to the sixth, seventh, and eighth cervical nerves and the first dorsal nerve. The four lower cervical nerves, however, and the first dorsal nerve, join to form the brachial plexus, and thus we find that the lower cervical ganglion and the inferior cardiac nerve rising from it are in connection with the brachial plexus. In addition to these should be mentioned the repeated anastomoses of the sympathetic with the vagus; the ganglia of the former are in communication with branches of the latter, both with several rising from its trunk, and with the

superior and inferior laryngeal nerves; and the vagus, again, is joined to the cervical nerves by a twig passing from it to the descending branch of the hypoglossal nerve. The cardiac plexus is abundantly connected by branches with the thoracic aortic plexus and the coronary plexuses of the heart. The occurrence of pain during the stenocardiac attacks in the *regions supplied by the cervical nerves* is thus explained by the communications existing between the cardiac plexus and the anterior divisions of the four upper cervical and first dorsal nerves, while the painful sensations radiating towards the *left arm* may be said to be due to the participation of the anterior division of the first dorsal nerve in the formation of the lower cord of the brachial plexus. That the pain is felt more frequently in the left arm than in the right is, perhaps, partly owing to the fact that the heart and aorta are situated more towards the left side of the body than the right, so that disease in the aorta is more likely to involve the nerves of the left side; it is also partly the result of the freer nervous anastomoses on the left side than on the right. The pain occasionally reaches the front of the chest, passing, probably, through the fasciculi which connect the dorsal spinal nerves with those of the brachial plexus. There are also radiating pains in the region of the diaphragm, which may be traced to the connection between the phrenic and the cardiac nerves, through the fourth and fifth pair of cervical nerves. The phenomena sometimes observed in the domain of the vagus—difficulty in swallowing, nausea, dysphonia—may be referred to the numerous anastomoses of the nerve with the sympathetic, especially to those between the cardiac plexus and the cardiac branches of the vagus. The communications of the cardiac plexus with the aortic and coronary plexuses explain the frequency of the stenocardiac attacks when the coronary arteries are ossified and contracted.

Sometimes we find the pain of angina pectoris confined to one part, such as the præcordial region, at other times radiating in the direction of the different nerve trunks. This depends to a great extent, next to simple mechanical conditions, on the intensity of the original irritation of the cardiac plexus. As in other neuralgic affections, we may assume that the number of nerves involved increases with the intensity of the pain. In some cases which were under our own observation some months we found that the more acute the primary pain in the præcordial region the

greater was its radiation, and that in cases of less intensity there was almost no radiation towards the left arm, the pain reaching only as far as the shoulder, while on the front of the chest it was but very slight.

The sensation of oppression and anxiety must be regarded as depending on the præcordial pain—at least such a relation seems to us to be more probable than the idea that it is the result of a change in the action of the heart. We have already, while discussing the præcordial pain, referred to such a theory, and may also here repeat that when there is obstruction of the circulation, such as occurs in the various valvular defects to a greater extent than in angina pectoris, this feeling of anxiety is absent.

Having discussed the neuralgic phenomena of the stenocardiac attack, we come now to the consideration of the *causes of the motor disturbance of the heart*.

As already stated, we cannot proceed on the assumption of excitement of any *single* system of cardiac nerves as the cause of these disturbances; we can only analyse the phenomena according to our present knowledge of the innervation of the heart, and so endeavour to show in what way the symptoms may be truly explained.

Three systems of nerves are concerned in the innervation of the heart—first, the *automatic* (ganglionic) system; secondly, the *vagus* system, which *moderates* the heart's action; thirdly, the *sympathetic* nervous system. Alteration in the action of any one of these may lead to the phenomena in question.

We refer first to the *automatic system* of cardiac nerves.

The heart continues its rhythmical action some time (in cold-blooded animals some hours) after being removed from the body. The sources of innervation necessary for this action are the groups of ganglia that lie embedded in the muscular tissue of the organ. Influences which arrest the functions of these ganglia, or of the muscular structures under their control, immediately cause the contractions of the heart to cease. An example of this is seen in the effect of different cardiac poisons introduced into the ventricular cavities of frogs, or when the entire heart is immersed in poisonous solutions. Landois* has shown that the disturbance of the automatic action of the ganglia by the direct influence of

* "Greifswalder medicinische Beiträge," ii. Band, 1864, p. 161-177.

certain poisons may be of two varieties—on the one hand stimulation, on the other depression, or even paralysis, of the heart's action. *Weak* solutions of various substances, brought into contact with the endocardium (in the frog), *irritate* the ganglionic cells lying underneath, and excite the heart to *increased activity*; *strong* solutions quickly paralyse the ganglia, and at once *arrest* the contractions of the heart.

In a way similar to that in which the above-mentioned physiological phenomena are brought about, the cardiac ganglia may be affected under pathological circumstances. If the rhythm of their action be disturbed by any cause, by abnormal *resistance* to the circulation of the blood (as in valvular insufficiency and atheromatous processes in the aorta), or if the blood-supply to the ganglia be inadequate (as in *contraction* or closure of the *coronary arteries*), or if, as is improbable, the ganglia become involved in diseases of the muscular tissues of the heart (such as myocarditis or fatty degeneration), an alteration in the action of the heart may take place, and that of one of two kinds: either the frequency of the contractions is *increased* when the above-named conditions give rise to *irritation* of the ganglia, or it is *decreased* when the influence is of a *paralysing* nature. The fact that in the stenocardiac attack this force is observed to be sometimes increased and sometimes diminished, is quite consistent with the assumption that this change may be connected with alteration in the function of the automatic ganglia; and as when poisonous solutions are injected into the endocardium the above-noticed contradictory phenomena follow, according to the degree of concentration, so one may suppose that the pathological irritation, according to its intensity, may be followed by increase or diminution of the heart's power.

In support of the theory that the ganglia are affected, perhaps in consequence of an inadequate blood-supply (as in aortic insufficiency and contraction of the coronary arteries), we may quote the experimental fact that after artificial closure of the coronary arteries the contractions of the heart are diminished in frequency, and in a short time cease, but return with perfect regularity when the ligature is undone. (v. Bezold.*)

The heart symptoms, at least in some cases, may be caused by disorder in the second of the above-mentioned cardiac sys-

* "Centralblatt für die med. Wissenschaften," 1867. No. 23.

tems of nerves—the *inhibitory* system of the *vagus* nerve. Thus, cases occur in which the pulse is retarded. Eichwald has observed many such, in which the pulse was at first slow, full, and hard, but subsequently, when the paroxysm had lasted somewhat longer, irregular, intermittent, or even imperceptible. These pulse-phenomena correspond to those observed when the *vagus* nerves in the neck are subjected to electric stimulation; thus we may assume that it is irritation of the *vagus* which gives rise to the slow pulse in the above-quoted cases of angina pectoris. In the majority of stenocardiac seizures, however, the pulse is accelerated, which points to the conclusion that the cardiac branches of the *vagus* are for the time paralysed, so that the restraining power usually exercised by them is diminished.

No pathological change in the *vagus* has yet been demonstrated in simple, uncomplicated angina pectoris. There is recorded, however, a case resembling this disease in many particulars, in which the *vagus* was found to be affected. The most striking symptom presented by the patient, who was in Skoda's clinique, and whose case is described by Heine,* was that the action of the heart was frequently *completely suspended* for some seconds, usually for a period that should have been occupied by 4-6 beats; he had also an indescribable sensation of anxiety like that in angina pectoris. On *post-mortem* examination, Rokitansky found the right phrenic nerve thrown up into dark blue knots and interspersed with chalky concretions. The *great cardiac nerve*, passing upwards from the plexus situated between the descending aorta and the pulmonary artery, united with the cords composing the sardiac plexus to form a black swelling of the size of a hazelnut just under the arch of those vessels, and was considerably thickened before passing into this enlargement. The *branches of the left vagus*, descending in front of the left bronchus to the pulmonary plexus, were found to be similarly involved in a dark-bluish tumour formed by a lymphatic gland.

There is also a form of angina pectoris occurring in diseases of the abdominal organs, which, on physiological grounds, we may regard as a reflex neurose of the *vagus*. This theory rests on the physiological fact that by irritating the sympathetic nerve in the abdominal cavity (as in Goltz's

* "Müller's Archiv für Physiologie," 1841, p. 236.

percussion experiment), one may arrest the heart's action in the diastole—exactly what follows direct irritation of the vagi in the neck. It was shown in the physiological part of this work that this effect on the heart is produced through nerves which excite the vagus by reflex action, which nerves are distributed in the trunk of the sympathetic and pass through the ramicommunicantes to the spinal marrow between the third and sixth vertebræ. After division of the vagi irritation of the abdominal sympathetic has no influence on the heart. In a similar way, it may be assumed that stenocardia, in diseases of the abdominal organs, is often due to a reflex disturbance of the heart by pathological irritation of the abdominal plexuses.

Finally, the phenomena of angina pectoris, with respect to the action of the heart, may also be explained by a condition of irritation in the *cervical or thoracic parts of the sympathetic*, which contain, as was formerly shown, the accelerator nerves of the heart.

The sympathetic may also be in another way concerned in these changes. Since the vasomotor nerves of the heart come from the sympathetic, any functional disorder of these is followed by a *change in the tone of the vessels*, and, therefore, by a *change in the blood pressure*, which may react on the heart in two ways: When the vasomotor nerves are *irritated*, the vessels *contract*; but when *paralysed*, *dilatation* of the vessels ensues. In the first case the action of the heart is rendered more energetic because the narrow peripheral channel offers an obstruction to the emptying of the heart, and *raises* both the *blood-pressure* in the aortic system and the frequency of the pulse; in the second case, on account of the diminished resistance to the circulation of the blood, the pressure in the aorta *becomes less*, the heart acts more feebly, and the frequency of the pulse is lowered. Cahen* long ago advanced the theory that certain cases of angina pectoris might be connected with the vasomotor fibres of the sympathetic. Some time after Landois,† and then Nothnagel,‡ recorded cases of angina pectoris, which they ascribed to *general arterial spasm*, frequently produced by the influence of cold in persons otherwise healthy; therapeutical agents

* "Archives générales de Méd.," 1863, Vol. ii., p. 564-570, and p. 696-698.

† "Correspondenzblatt für Psychiatrie," 1866.

‡ "Angina pectoris vasomotoria," "Archiv für klinische Medicin," 1867, Band iii., p. 309.

which relieved this state of spasm (such as irritation of the vessels of the skin, application of warmth) also arrested these attacks.

Pathological anatomy furnishes us with only one, but a very important, fact, which goes to support the theory that the sympathetic is concerned in the production of this disease. In the body of a man, 45 years of age, who died in an attack of angina pectoris, from which he had for a long time suffered, Lancereaux* found pathological alterations in the cardiac plexus. We quote the report literally:—

“A l'autopsie on constata l'existence d'une lésion de l'aorte. Entre les deux orifices des artères coronaires rétrécies au point de permettre à peine l'introduction d'un stylet, se trouve une plaque saillante de plusieurs centimètres d'étendue, à rebords festonnés, et composée en grande partie de tissu conjonctif de nouvelle formation. Situé entre la couche interne et la couche moyenne le néoplasme paraît contenir dans son épaisseur de fines arborisations; la tunique externe de l'aorte était le siège d'une vascularisation anormale extrêmement riche. *Le plexus cardiaque participait à cette vascularisation*, et quelques uns de ses filets se trouvaient compris dans une sorte de gangue ou de plasma appliqué à sa tunique externe épaissie. L'examen microscopique des filets nerveux et des ganglions montra d'une façon positive que des nombreux noyaux ronds se trouvaient interposés sous forme d'amas entre les éléments tubuleux qu'ils comprimaient plus ou moins; la portion médullaire de ces éléments était d'ailleurs grisâtre et grenue.”

We also, in several cases, have made a therapeutical trial of galvanisation of the sympathetic in angina pectoris, but have not been rewarded by any very evident good result. On the other hand, Cordes† reports that he produced very manifest improvement in one case by galvanising the left sympathetic. This case, which was of the kind formerly spoken of as angina pectoris *vasomotoria*, is specially interesting, as the left sympathetic was painful and evidently irritated, and there was a persistent dilatation of the left pupil.

To sum up, we would state that the symptoms in angina pectoris may be referred to various causes, some of which

* “De l'altération de l'aorte et du plexus cardiaque dans l'angine de poitrine,” *Gaz. méd.*, 1864, p. 432.

† Cordes, “*Deutsches Archiv für klin. Med.*,” 1874, Bd. xiv., p. 141. v. Hübner, also, has got the most favourable results from galvanising the sympathetic in one case of angina pectoris. “*Archiv für klin. Med.*,” 1873, Bd. xii.

may be external to the heart; that probably all the cardiac nerves, and the ganglionic apparatus of the heart, co-operate to a greater or less degree; and that the variable character of the phenomena observed in different cases may be traced to the greater or less share taken by the different nerves forming the cardiac plexus. *The sympathetic is probably most considerably involved*, as it takes the principal part in the formation of the cardiac plexus.

IX.—ADDISON'S DISEASE.

Bronzed Skin.

In the great majority of cases of Addison's disease degeneration of the suprarenal capsules has been found, and the inference has been, with perfect justice, made, that that degeneration must be, in some way, connected with the clinical symptoms of the disease. The objections, that diseases of the suprarenal capsules occur without bronzing of the skin, and that instances have been observed of complete absence of these bodies in men who were quite healthy during life,* do not weaken the above theory. Another question is whether the only cause of bronzed skin is to be sought in degeneration of the suprarenal bodies, and whether the latter is the primary or the secondary affection.

After Addison had drawn attention to the suprarenal capsules, physiologists soon attempted to determine experimentally what the hitherto unknown function of these bodies might be. The results of these researches, however, did not support the assumption that the cause of Addison's disease is to be found in affection of the suprarenal glands. Brown-Séquard† certainly observed that death occurred very quickly on extirpating the suprarenal capsules in animals; further, after the extirpation, he noticed an accumulation of pigment in the blood, and from these facts concluded that the suprarenal bodies are organs whose function it is to separate and to destroy pigment. But all other authors who have repeated these experiments on many different kinds of animals, contradict those statements. Gratiolet,‡ Philip-

* Martini, "Comptes rendus," 1856, Tome xliii., p. 1052. Kent Spender, "British Med. Journal," 11 Sept., 1858. Stedmann, "Guy's Hosp. Reports," viii., 1863, p. 1.

† "Comptes rendus," 1856, Tome xliii., p. 422 and 904; "Comptes rendus," 1857, Tome xliv., p. 246; and Tome xlv., p. 1036.

‡ "Comptes rendus," 1856, T. xliii., p. 468.

peaux*, Berruti and Perusino†, Harley,‡ Chatelain,§ Schiff,|| &c., have shown that if death occur soon after the extirpation it is merely the result of the severity of the operation, because, under favourable circumstances, they have kept alive many animals, both those possessing pigmented tissues and others that were albinos, weeks and even months after cutting out the suprarenal capsules, while, at the same time, no increase of the pigment of the skin was observed, nor, after death, any anomaly of pigmentation in the internal organs.

Chemical examination of the suprarenal bodies has furnished us with no certain information regarding their normal functions, or the pathogeny of Addison's disease (Vulpian,¶ Virchow,** Arnold††).

Virchow‡‡ has pointed out the resemblance of the colouring matter found in the normal suprarenal capsules to that of the rete Malpighii, but in how far this normal pigmentation is connected with the staining of the skin is still undetermined; on the other hand, little value is to be put on the statement that in negroes the suprarenal capsules are relatively very large (Casson)§§, an observation not confirmed by Cruveilhier.|||| Opposed to these negative results of experimental research, and partly before physiologists had become acquainted with the over-estimated importance of the function of the suprarenal bodies, another theory regarding the origin of Addison's disease had been put forward, according to which it is of a secondary character, *depending on an affection of the nervous system, especially of the great abdominal plexuses of the sympathetic.*

This view evidently originated in observation of the relation subsisting between the nerves of the suprarenal capsules and the abdominal plexuses of the sympathetic. Thus, the ganglion semilunare sends a considerable number of twigs to the suprarenal bodies, and these form there a close network, which is, as Virchow ¶¶ discovered, richly supplied with

* "Comptes rendus," 1856, T. xliii, p. 964 and 1155; T. xliv., p. 396.

† "Canstatt's Jahresbericht," 1857, iv., p. 265.

‡ "British and Foreign Med. Chir. Review," 1858, vol. xxi., p. 204 and 498.

§ "De la peau bronzée, ou Maladie d'Addison," Thèse, Strassbourg, 1859.

|| "Union Médicale de Paris," 1863, No. 61, p. 346.

¶ "Comptes rendus," 1856, Tome xiii., p. 664; and Tome xiv., 1857, p. 340.

** "Archiv für Path. Anat.," 1857, Bd. xii., p. 481.

†† "Archiv für Path. Anat.," 1866, Bd. xxxi., p. 64.

‡‡ "Die krankhaften Geschwülste," Bd. ii., p. 695.

§§ See Virchow, "Die krankhaften Geschwülste," Bd. ii., p. 695.

|||| See "Schmidt's Jahrbücher der Medicin," Bd. cxix., p. 239.

¶¶ "Virchow's Archiv," 1857, Band. xii., p. 483.

ganglia. These anatomical considerations and the changes—which we shall discuss further on—lately found in the abdominal plexuses of the sympathetic in a great number of cases, have tended to strengthen the theory that Addison's disease is intimately connected with structural change in the sympathetic. The great majority of authors, including, most recently, Headlam Greenhow,* are inclined to this belief. We have not, however, any very good *physiological* reason for this, for we can just as seldom produce the symptoms of Addison's disease by extirpating the abdominal plexuses of the sympathetic as by excising the suprarenal capsules. The results of a number of these experiments are given in the physiological part of this essay.

While experimental physiology thus seems rather opposed to the theory that this disease is dependent on an affection of the abdominal sympathetic, pathological anatomy has comparatively lately furnished us with certain observations which point with some show of probability to this conclusion. The number of cases in which morbid changes have been found in the sympathetic is not small; in many of these, however, the examination appears to have been incomplete, and, with respect to some, it may be questioned whether the structural alterations observed possess any real pathological value, and even when they can claim such value, whether they should be regarded as primary, or only as secondary affections. Such as they are, we now proceed to quote these cases in detail:—

1. Queckett † found fatty degeneration of the solar plexus in one case.

2. Recorded by Monro.‡—A woman, 40 years of age; both suprarenal capsules degenerated, and adherent to the surrounding parts—the right enlarged to four times its proper size. *The sympathetic nerves from the small splanchnic, and some of the ganglia of the solar plexus, swollen and of a reddish colour (hyperæmic); the left suprarenal body had preserved its normal size and position, and its nerves were less injected.*

3. Recorded by Washington Lovegrove.§—An engineer, 32 years of age.—Autopsy performed by Wilks. Both supra-

* "Lancet," 1875, Vol. i., p. 327, 361, 395, 429, 463, 532.

† The particulars of this case are not given in any of the books or publications to which we have had access.

‡ Monro, "Assoc. Med. Jour.," 1856, p. 848.

§ Washington Lovegrove, "Med. Times and Gaz.," 1858, 17th July.

renal capsules degenerated into albuminous, chalky masses, adherent to the surrounding tissues—the right considerably enlarged. *The semilunar ganglia were healthy, but the branches which they gave to the diseased glands were completely atrophied.*

4. Recorded by F. J. J. Schmidt.*—A servant girl, 16 years of age.

Section, by Dr. Boogard.—The suprarenal capsules slightly enlarged; *the sympathetic in the neighbourhood of the abdominal aorta to a high degree atrophied.*

5. Recorded by van Andel.†—A woman, 30 years of age.

Section.—Both suprarenal capsules totally changed by tuberculous degeneration. *Microscopic examination revealed the presence of atrophy of the sympathetic, and of the solar plexus, with almost entire disappearance of the medullated cells, and brown pigmentation of the ganglionic cells, in which the nucleus could be distinguished only by means of its nucleolus.*

6. Recorded by Gull.‡—A man, 31 years of age.

Section, by Wilks.—Both suprarenal capsules transformed into large albuminous masses. The surrounding tissues were included in this new formation, so that *the right semilunar ganglion and its nerves were completely embedded in it, whilst the left was free, only its branches being enveloped by and lost in the mass.*

7. Recorded by Habershon.§—A bookbinder, 18 years of age.

Section.—Caseous and calcareous degeneration in both suprarenal capsules, the left enlarged. *The left semilunar ganglion lay close to the left suprarenal capsule, and several large branches from it were firmly embedded in the solid new formation; microscopic examination, however, showed no change in the ganglion cells.*

8. Recorded by v. Recklinghausen.||—A woman, 40 years of age.

Section.—The normal structure of the suprarenal capsules quite lost, but replaced by a grey tissue well supplied with blood, the products of chronic inflammation which had undergone fatty metamorphosis. *The celiac ganglion was injected, but nothing abnormal was found when it was subjected to ex-*

* F. J. J. Schmidt, "Archiv für die holländischen Beiträge," Bd. ii., p. 166.

† van Andel, "Nederl. Tijdschr. v. Geneesk," vi., p. 200 (April, 1862).

‡ "Med. Times and Gaz.," 1863, 24 Jany.

§ Habershon, "Lancet," 1864, 5 March, p. 269.

|| v. Recklinghausen, "Deutsche Klinik," 1864, No. 8, p. 78.

amination. The nerves of the sympathetic, including those passing to the suprarenal capsules, presented nothing abnormal beyond being very full of blood.

9. Virchow* states that he has observed, in an individual who had suffered from cancer of the œsophagus and bronzed skin, a hyperplastic hæmorrhagic swelling of the suprarenal capsules, accompanied by thickening of the solar plexus.

10. Recorded by Headlam Greenhow.†—A man, 32 years of age.

Section.—Both suprarenal capsules enwrapped in thick fibrous tissue—the right considerably enlarged, the left smaller, and consisting of fibrous tissue interspersed with caseous and chalky masses. The nerves from the semilunar ganglion entering the affected gland were at least twice as large as usual, but showed under the microscope only an increase of the fibrous sheath of the nerve bundles.

11. Recorded by Meinhardt.‡—A man, 52 years of age.

Section.—Both suprarenal capsules degenerated. The microscopic examination (by Luschka) showed that the medullary part of the gland had the appearance of a tuberculous mass consisting of molecular detritus and globules of fat. Nerve-tubules and ganglionic cells were completely absent.

12. Recorded by Bartsch.§—A clerk, 48 years of age.

Section, by Perls.—Both suprarenal capsules the seat of caseous degeneration. The semilunar ganglia were of normal size, embedded in a quantity of loose fatty connective tissue, greyish-red in colour, and presented a regular surface when cut. Microscopic examination showed the presence of whole ganglionic cells almost completely filled with small brown fatty molecules, only a few cells preserving a distinct nucleus. After treatment with acetic acid, the oblong nuclei in the pale grey fibres of Remak were decidedly fewer than normally, the bulk of the ganglia at most parts consisting of a fibrous connective tissue interspersed with fine, strongly-refracting molecules, and here and there with long narrow nuclei. The proper nerve fibres showed nothing striking, either in external appearance or in their distribution.

13. Recorded by Sanderson.||—In the case of a woman, the

* Virchow, "Die krankhaften Geschwülste," Bd. ii., p. 697.

† Greenhow, "Path. Transact.," xvii., p. 307.

‡ Meinhardt, "Wiener med. Presse," 1866, No. 1-4 and 7-9.

§ Bartsch, "De Morbo Addisonii," Inaugural Dissertation, Königsberg, 1867.

|| Sanderson, "Med. Times and Gaz.," 1868, Oct. 31.

semilunar ganglia, and the nerves connecting them with the suprarenal capsules, were enclosed in adenoid tissue. This tissue also surrounded and united the suprarenal bodies—which were fatty in structure, though still showing distinct remains of the cortical substance—to the neighbouring organs.

14. Recorded by M. Wolff.*—A merchant's apprentice, 16 years of age.

Section.—Both suprarenal capsules enlarged to three times their natural size, and degenerated; microscopically, not a trace of the normal structure of these organs could be seen. *The nerves of the solar plexus, and the semilunar ganglia with the nerves passing from them to the suprarenal bodies, were surrounded by a firm envelope of connective tissue. On some of the nerves from the solar plexus, before they enter into the corresponding semilunar ganglion, were ampulliform swellings.*

Microscopic examination of the semilunar ganglia showed that the greater number of ganglionic cells were intact and had a distinct nucleus, only a few of them containing some fatty molecules. In the nucleated fibres of Remak in the ganglia themselves nothing abnormal was observed. *The increase in the amount of connective tissue, however, in the ganglia was very striking. This tissue was fibrous, and here and there provided with spindle-shaped cells.—The ampulliform swellings, situated on some of the nerves of the solar plexus, just before entering into the corresponding semilunar ganglia, were seen, on microscopic examination, to be small ganglia containing ganglionic cells. In these, as in the large semilunar ganglia, the hyperplasia of connective tissue between the ganglionic cells was well marked. Examination of individual nerves of the solar plexus demonstrated no other irregularity in the nerves themselves, but the already mentioned thickening of the neurilema which, in cross section, showed itself by numerous processes passing in between the nerve bundles. The principal element of the process going on in the ganglia and nerves was thus found to be development of connective tissue, but of such a nature that it did not produce atrophy either in the ganglionic cells or in the nerve filaments.*

15. Recorded by Kuhlmann.†—A man, 33 years of age.

Section, by Rindfleisch.—The suprarenal capsules shrunken and full of tuberculous material. In the adhesions of the

* M. Wolff, "Berliner klinische Wochenschrift," 1869, Bd. 17, 18.

† Kuhlmann, "Berliner klin. Wochenschrift," 1869, No. 45.

capsules were found, on microscopic examination, two large nerve trunks, the fibres of which had become the seat of *fatty degeneration*.

16. Recorded by A. Fränkel.*—A man, 30 years of age.

Section.—Right suprarenal body almost double its normal size, degenerated, and containing some thick pus; the left rough, and of the size of a walnut, of the same structure. *On this (the left) side the suppurative softening involved the outer part of the solar plexus, and formed in it an abscess of the size of a cherry.* No microscopic examination was made.

17. Recorded by Burresi.†—A man, 43 years of age.

Section.—Degeneration of both suprarenal capsules. *The semilunar ganglia considerably enlarged, especially the left; the nerve trunks forming the solar plexus thickened; the ganglionic cells (under the microscope), especially on the left side, more granular and indistinct than usual, without a trace of a nucleus; the neurilema hypertrophied.* Further examination showed that *the whole sympathetic nervous system was injected and swollen, that both upper cervical ganglia, especially the left, were increased in size, more cylindrical than normal, and prolonged and tapering in a downward direction; that the neurilema was hypertrophied, and that the enclosed medullary substance was less in quantity.* Microscopic examination revealed the presence of both old and recent effusions of blood in the ganglia and nerve trunks.

18. Recorded by Southey.‡—A woman, 36 years of age.

Section.—The capsules of the suprarenal bodies transformed into a caseous mass. *The semilunar ganglia similarly affected.*

19. Recorded by H. M. Tuckwell.§—A woman, 31 years of age.

Section.—The suprarenal bodies degenerated; the left united to the liver by a mass of connective tissue. *The nerves entering the suprarenal plexus, the semilunar ganglion, and the great splanchnic nerve, were enveloped in the above-mentioned mass of connective tissue.*

20. Recorded by Trübiger.||—An apprentice, 17 years of age.

* A. Fränkel, "Ein Fall von Addison'scher Krankheit," Inaug. Dissert., Berlin, 1870.

† Burresi, "Lo Sperimentale," xxv., 6.) (Anno. xxii., 1870), p. 521.

‡ Southey, Pathological Society of London. Sitting of 19 Decbr., 1871.

§ Tuckwell, "St. Barthol. Hosp. Rep.," vii. (1871), p. 73.

|| Trübiger, "Archiv der Heilkunde," 1874, Bd. xv., p. 417.

Section.—The suprarenal capsules the seat of cheesy degeneration. A *small-celled infiltration* was found in the *great ganglia* round the left suprarenal body, and in these bodies themselves. *The ganglionic cells were unchanged.*

We will now quote some other cases, in which *no change was found in the sympathetic.*

1. Recorded by Martineau.*

Section.—Both the suprarenal capsules enlarged, and adherent to the surrounding tissues. *The nerves of the celiac plexus, and the ganglia of the solar plexus, were examined most carefully, and showed no change.*

2. Recorded by Child.†—A woman, 37 years of age.

Section, by Tuckwell.—Both suprarenal bodies enlarged, and infiltrated with tubercles. *No lesion in the nerves supplying the suprarenal glands.*

3. Recorded by D. Williams.‡—A woman, 46 years of age.

Section.—The suprarenal bodies considerably enlarged and degenerated. *The sympathetic nerves of the small splanchnic and the ganglia of the solar plexus were normal.*

4. Recorded by Chatin.§—A man, 46 years of age.

Section.—Both suprarenal capsules the seat of tubercular degeneration. *In the semilunar ganglia no fatty metamorphosis could be detected.*

5. Recorded by van den Corput.||—A woman, 30 years of age.

Section.—The left suprarenal body tuberculous, the right entirely atrophied, *the nerves of the plexuses apparently normal.*

6. Recorded by Heslop.¶—A man, 21 years of age.

Section.—Both suprarenal capsules the seat of cheesy degeneration. *No change in the sympathetic nerves and ganglia.*

7. Recorded by Schüppel.**—A farm servant, 34 years of age.

Section.—*The sympathetic was perfectly normal.*

* Martineau, "De la Maladie d'Addison," Paris, 1864.

† Child, "Lancet," 18 Feb., 1865, p. 176.

‡ Williams, "Brit. Med. Journal," 9th February, 1867.

§ Chatin, "Gaz. méd de Lyon," 1867, p. 257.

|| Van den Corput, "Journ. de Bruxelles," xvii., p. 573. Dsch, 1868.

¶ Heslop, "Lancet," 1870, No. 23.

** Schüppel, "Archiv der Heilkunde," 1870, xi., p. 87.

8. Recorded by Rossbach.*—A woman, 62 years of age, who had suffered from Addison's disease, complicated with scleroderma.

Section.—Both suprarenal capsules normal. *No changes* were observable in the thoracic or abdominal parts of the *sympathetic*, or in the solar plexus.

9. Recorded by H. Wolff.†—A man, 50 years of age.

Section.—Suprarenal bodies normal; *nothing abnormal in the sympathetic.*

10. Recorded by Krause.‡—A man, 20 years of age.

Section.—*Semilunar ganglia normal.*

11. Recorded by Eppinger.§—A woman, 40 years of age.

Section.—*No change in the ganglionic cells of the abdominal ganglia of the sympathetic.*

12. Recorded by Pye Smith.||

Section.—Both suprarenal capsules degenerated; the *ganglia and the solar plexus*, however, *unaffected.*

Thus the results of examination of the sympathetic still remain antagonistic to each other, at one time negative, at another positive. But even should the positive evidences accumulate in the future, or if it be shown that the changes in the plexuses of the sympathetic are primary, and those in the suprarenal capsules secondary phenomena, the question would still be *how* the symptoms of Addison's disease are caused by such changes—a question towards the solution of which we have not advanced one step.

X.—DIABETES MELLITUS.

In the physiological part of this essay mention is made of the well-known facts that in animals diabetes mellitus may be produced by wounding the fourth ventricle of the brain, by making incisions in the spinal marrow at various parts from the medulla oblongata downwards to the level of the lumbar vertebræ, and by injuring the cervical and upper thoracic ganglia of the sympathetic. It was further stated

* Rossbach, Virchow's "Archiv," 1870, Bd. l., p. 566, and Bd. li., p. 100.

† H. Wolff, "Ein Fall von Broncekrankheit," Inaug. Dissert., Berlin, 1872, p. 25.

‡ Krause, "Sitzungsberichte des Vereins der Aerzte in Steiermark," ix., p. 22 (1871-72).

§ Eppinger, "Böhmisches ärztliches Correspondenzblatt," 1875, No. 29, p. 259.

|| Pye Smith, Virchow's "Archiv," 1875, 65th Band., p. 502.

in detail that all these experimental procedures were followed by injury (paralysis) of the vaso-motor nerves of the liver, which rise in the neighbourhood of the arch of the fourth ventricle of the brain, traverse the cervical and dorsal parts of the spinal marrow to the fourth or fifth dorsal vertebra, join the sympathetic through the ramicommunicantes, pass downwards towards the liver, and eventually enter it as the hepatic plexus.

Injury to these vaso-motor nerves, no matter at what part of their course, produces a paralytic dilatation of the vessels of the liver; this causes an increased flow of blood, and thus an augmentation in the quantity of sugar is formed, which then enters the circulation, and finally the urine. As we have formerly gone over these details, we have here only to answer the question whether diabetes in man is in any way dependent on disease of the sympathetic nervous system.

This is undoubtedly the case in those forms of the disease which are analogous to diabetes in animals after injury to the central parts of the nervous system. To this category belongs diabetes from injury or disease of the brain involving the fourth ventricle and the parts near it, of which class of cases there are many recorded examples. Probably, also, many forms of *toxic* diabetes (as by carbonic acid poisoning), and the rare forms of intermittent diabetes (the analogues of other intermittent neuroses) may be due to some affection of the vaso-motor nerves.

A considerable quantity of sugar has lately been found in the human urine (Braun*) in certain cases of neuralgia of the sciatic nerve; this condition seems to resemble those cases in animals in which diabetes followed division of the sciatic nerve. In one case of inveterate sciatica of the right side we occasionally found sugar to the extent of about one per cent., the quantity of urine passed daily being 3,000 ccm., and the specific gravity 1023. The sugar disappeared when the sciatica was cured, the specific gravity sinking to 1008.

While discussing hyperidrosis unilateralis we mentioned that in three recorded cases of diabetes there were signs of disordered function in the sympathetic, usually increased perspiration on one side; in one case there was also contraction of the pupil. Till we have further observations on this point it must remain undecided whether there is any pathological connection between hyperidrosis and diabetes.

* Braun, "Lehrbuch der Balneotherapie" (Berlin, 1868), p. 343.

We possess no pathological facts that would warrant the assumption that the sympathetic takes part in causing other varieties of diabetes. Nevertheless, we would mention that in a case of this disease, complicated with atrophy of the pancreas, Klebs and Ph. Munk* found the semilunar ganglia atrophied, whilst the hepatic nerves running along with the hepatic artery remained perfectly unaffected. Whether the atrophy of the solar ganglion has any relation to the atrophy of the pancreas or to the diabetes is unknown. The same authors noticed also that in dogs, after partial extirpation of the solar ganglion, diabetes appeared and partly passed off again, traces of it being still present, however, till death occurred (1-2 weeks). At the *post-mortem* examination of the last case it was noticed that the nerve elements were degenerated.

XI.—HYPERÆSTHESIAS OF THE SYMPATHETIC SYSTEM.

1.—*Hyperæsthesia of the Mesenteric Plexus.*

(Enteralgia, Enterodynia, Colic.)

Those affections of the intestinal canal and the parts connected with it, usually known as Enteralgia or Colic, which Willis† seems to have been the first to consider of a neuralgic nature, have been for some time regarded by most authors as neuroses of the sympathetic; and as their seat has been placed in the mesenteric plexus, they have been described as *Hyperæsthesia plexus mesenterici* or *neuralgia mesenterica* (*mesaraica*). This refers both to the ordinary colic (also known as rheumatic, hysterical, &c.), and also to that well-marked form called *Colica Saturnina* (*colica pictonum* of the older writers), which is etiologically and clinically almost typical; it includes also some endemic forms of colic, apparently almost identical with lead colic (colic of Poitou, Madrid, Devonshire, Cayenne, “*colique végétale*,” &c.).

De Haen and Vanstrostwyk ascribe lead colic to a diseased condition of the abdominal ganglionic system. The same view is taken by Andral, Grisolle, and Ranque, who further hold that the spinal marrow as well as the sympathetic is concerned in the process. On the other hand, Astruc and Sauvages believe that it rises purely from some spinal cause,

* “*Tageblatt der Naturforscher-Versammlung zu Innsbruck*,” 1869, p. 113.

† Willis, “*Op. omn.*,” ed. Geneva, T. ii., p. 323.

while different authors have absolutely denied its neurotic nature, and traced it to other local anatomical changes in the intestinal canal or in the abdominal coverings.

Tanquerel des Planches, who has had unusual opportunities of studying saturnine diseases, states most decidedly that the seat of lead colic must be sought exclusively in the *sympathetic system*. Tanquerel appealed to the belief most common amongst the physiologists and pathologists of his time, Bichat, Brachet, Andral, Jolly, &c., that the sympathetic system constituted the *motor and sensory centre* for the vegetative organs of the body.* “Should it ever be discovered—which is very improbable—that nerve fibres from any other part than the ganglionic nervous system supply motor and sensory power to the abdominal organs, in them we may expect to find the cause of colic; but till such a discovery is made we can recognise no other seat for this affection.”

From this standpoint—which was quite a just one considering the state of knowledge regarding physiological function at that time—Tanquerel also disputed the theory adopted by Andral and others, that the spinal cord had also something to do with it; the latter, according to him, takes part in producing colic only when it is accompanied by paralysis and arthralgia saturnina. “Colic has its seat in the sympathetic, and nowhere else.”

Amongst the reports of 49 autopsies, recorded by Tanquerel, there is one which possesses for us a special interest, because in it are mentioned considerable changes in the sympathetic. We will quote it in Tanquerel's own words.†:—

“In the body of case 25 the ganglia of the sympathetic in the abdominal cavity were twice, some of them three times, their normal size, as calculated by comparison with the ganglia in two other cases. These ganglia were externally and internally of a yellowish-grey colour, but were not perceptibly indurated. Nothing of special importance was found in the plexuses. The ganglia of the thorax and neck appeared relatively not to have become so large as those in

* Tanquerel des Planches, “*Traité des maladies de plomb ou saturnines*,” Paris, 1839. The following quotation is from a German translation, published by Frankenberger in 1842, p. 208. Tanquerel holds that the seat of lead colic is not exclusively in the mesenteric plexus, but according as certain plexuses are specially involved the colic appears as epigastric, umbilical, hypogastric, renal, originating in the coeliac, mesenteric, hypogastric, or renal plexus.

† L. c., p. 200.

the abdomen. The other nervous ganglia showed nothing further which might distinguish them from those of the two other individuals with whom comparison was made."

In all the other cases the report concerning the sympathetic was negative. Andral,* Gendrin and others, could discover no alteration of the normal structure in this or in the other parts of the nervous apparatus. Tanquerel feels himself forced to regard the changes found in case 25, not as anatomical *causes*, but as *results* of the colic.† The other local changes found in lead colic are both inconstant in occurrence and unimportant,‡ and, moreover, their pathogenetic significance is limited by the circumstance that in those cases in which *post-mortem* examination has been made death was not, as a rule, caused by the lead colic, but by some complication. We have no further record of disease in the sympathetic or any other parts of the nervous system in lead colic, with the exception of one case described by Kussmaul and Maier.§ It occurred in the person of a painter, who had long suffered from chronic lead poisoning, and who died suddenly in an attack of colic. Besides chronic catarrh of the whole intestinal tract, there was fatty degeneration and wasting of the pancreas, and slight fatty infiltration of the stomach, especially towards the pyloric end; in the jejunum, ileum, and upper part of the colon there was atrophy of the mucous membrane, both of the stroma and of the glands and villi. There was increased development of the sub-mucous coat of the stomach and intestines by hypertrophy of its areolar tissue and thickening of the *adventitia capillaris*, the meshes of this layer being also filled with fat; the muscular coat of the intestine, especially the small intestine, was the seat of adenoid degeneration. *In the sympathetic many of the ganglia, especially the celiac and upper cervical, were indurated, the septa of connective tissue being hypertrophied and hardened.* Kussmaul and Maier believe that the chronic dyspepsia which

* Andral, "Clinique médicale," 3 ed., Tome ii., p. 229.

† Tanquerel, L. c., p. 201.

‡ Amongst his 49 cases Tanquerel found "balling" of the intestines 16 times, apparently accompanied by contraction; hypertrophy of Brunner's glands seven times; slight swelling of Peyer's patches three times; thick layers of exuded mucus on the mucous membrane four times; deep-seated softening five times; a *completely normal state of the canal* 20 times. Other good observers (Andral, Copland, Louis, Stokes, &c.) could find no pathological changes in the intestine.

§ Kussmaul and Maier, "Deutsches Archiv für klinische Medicin," 1872, ix., p. 283.

exists during life, the anæmic pallor, the deficient nutrition, and perhaps, also, the habitual constipation (atony of the intestinal muscles and diminished secretion from the intestinal glands), are dependent on the above-mentioned pathological phenomena. They, however, regard the theory concerning the influence of the sympathetic in bringing about lead colic as wanting further confirmation.

As regards the other forms of neuralgia mesenterica, we possess no important pathological anatomical facts, with the exception of one case of endemic colic observed by Dr. Ségond, in Cayenne, in which some ganglia and nerves of the sympathetic appeared to be hypertrophied, harder than usual, and of an abnormal colour.*

Under these circumstances we must confine ourselves entirely to the results of clinical observation, and it is questionable whether we are thus provided with very strong evidence of the sympathetic origin of colic. At the present day we need not discuss the doctrine, believed in by Tanquerel des Planches and many other physiologists and pathologists of his time, that the sensory and motor centre for the intestinal viscera is to be found only in the ganglia of the sympathetic. We know, on the contrary, that the sensorium commune in men is exclusively cerebral—*i.e.*, that sensory impressions are felt only in the brain, and that also the movements of the vegetative organs are in various ways controlled and modified by the cerebro-spinal nervous centres, as has been proved by numberless experiments and pathological observations relative to the stomach, intestines, ureters, bladder, uterus, vasa deferentia, &c. Such a statement as Tanquerel des Planches' would now be an anachronism. If we keep in view the neuralgic nature of the group of symptoms known as enteralgia or colic, the only important subject for investigation is *concerning the peripheral course* of the irritating action; whether—to express it more clearly—this is conveyed to the sensory centre by *sympathetic* or exclusively by *cerebro-spinal* afferent fibres. In the first case the sympathetic would have to be regarded as entirely a sensory nerve, the analogue of the sciatic nerve in sciatica, or of the trigeminus in prosopalgia.

Romberg, who describes the "hyperæsthesias of the sympathetic nerve" as a separate division of the second form of neuralgia ("hyperæsthesia from irritation of the central appa-

* Ségond, "Essai sur la neuralgie du grand sympathique, maladie connue sous les noms de colique végétale, de Poitou," &c. Paris, 1837.

ratus"), and includes under that designation not only *cœliac*, *mesenteric*, *hypogastric*, *spermatic*, and *uterine neuralgia*, but also *cardiac neuralgia* (*angina pectoris**), writes in the following way of the general characteristics belonging to this group of diseases †:—

"The hyperæsthesias of the sympathetic nerve are distinguished by certain peculiar features, which depend on the physiological function of that portion of the nervous system; first, the exciting of reflex action in the muscles, both voluntary and automatic. The impressions made on the sensory fibres of the sympathetic in health rarely reach our consciousness, but at once bring about reflex actions. In the hyperæsthesias, however, conduction takes place in both directions, and so not only perception of the sensation follows, but also contraction of the muscular fibres, whether in the heart, intestinal canal, excretory ducts of the glands, or in the walls of the abdomen, &c. Next to reflex action, the nervous energy that governs nutrition is more affected than in the hyperæsthesias of the cerebro-spinal nerves. The so-called vegetative processes (secretion, and even the circulation) are disturbed."

If we apply these general expressions to the affection specially under consideration, we observe in colic (both in the ordinary and in the saturnine forms) a number of abnormal motor phenomena, which have been understood to be dependent on reflex action. These are the partial, spastic contractions of the intestine, which in some cases, especially of lead colic, are not only subjective sensations, but may be felt by the hand, and which have their seat chiefly in the lower part of the canal, the *cœcum* and *colon*; the spasmodic contraction of the *sphincter ani* also, so often observed by Tanquerel, has been regarded as of this nature. Other concomitant symptoms of colic (nausea, vomiting, painful micturition, dragging upward of the testicle, &c.), have been similarly explained as reflex actions connected with the stomach, urinary organs, *cremasters*, &c.; more particularly the tension and stiffness of the abdominal walls during the attack have been thought to be of reflex origin.

This theory, however, is clearly founded on the error that

* Romberg, "*Lehrbuch der Nervenkrankheiten*," 2te Aufl., 1851, Bd. i., p. 14, p. 141-170.

† *Ibidem*, p. 142.

in colic we have to do with a genuine neurose of a sensory nerve, that the essential element in it is disturbance of sensation, and that the motor phenomena are merely accidental or secondarily dependent on it. Such a doctrine is as untenable in this disease as in angina pectoris or hemicrania. When we, in the ordinary but inaccurate classification of nervous diseases, include colic among the neuroses or hyperæsthesias, we act in conformity with the maxim, "*A potiori fit denominato*," inasmuch as we give most prominence to the symptom which is subjectively the most urgent, or practically the most important—the pain. *In fact, however, colic is just as little a hyperæsthesia of the mesenteric plexus as angina pectoris is of the cardiac plexus; it is rather a compound sensor-motor neurose, i.e., the same morbid actions which cause the irritation may at the same time produce the anomalies of motion through the motor nervous apparatus and muscular fibres.* This is most obviously the case in lead colic. From many experiments on animals, and from the results of chemical research in men (conducted by Tanquerel des Planches, Meurer, Devergie, Orfila, Chevallier, Chatin) we know that the lead is deposited most abundantly in the muscular system; we know, further, that it acts locally in a high degree as a stimulus to contraction in the unstriated muscles, either directly or through the medium of the intramuscular nerve-terminations; and to this are, for the most part, due the well-known astringent, styptic, and hæmostatic properties of the preparations of lead. Thus, nothing is more probable than that the lead deposited in the walls of the intestines stimulates the unstriated muscular fibres to contract, and so gives rise to the partial spasmodic constrictions of the intestinal tube. This may be the case in the smooth muscular fibres of the œsophagus, stomach, ureters, bladder, urethra, cremasters, &c. As regards the theory that the hardness and tension of the walls of the abdomen arise from reflex contraction of the abdominal muscles, it may be stated that it has no physiological warrant; at least we are not aware that any one has ever succeeded in producing reflex contractions of the abdominal muscles through the sensory visceral nerves. Moreover, these symptoms are neither constant in occurrence nor proportionate to the intensity of the pain; they may, as Romberg justly observes, be quite absent when the pain is very acute, a circumstance which seems to us to tell against the assumption of any reflex connection between the symptoms. We leave it an open question

whether the tension of the abdominal coverings in lead colic is perhaps the result of a direct excito-motor action of the lead, the possibility of which, even when voluntary muscles are involved, being unquestionable, or whether it should be reckoned a consensual movement of co-ordination, occasioned by the painful contraction of the lower part of the intestine. We would only protest against the readiness with which the term "reflex action" is used, without any just cause or physiological reason, as a handy explanation of any phenomenon in the domain of the sympathetic.

There is another train of symptoms which appears to us to be of greater importance and less doubtful significance, and to point to a *generally disordered state of the circulation*, and especially to a *diminution in the force of the heart's action* during the colicky seizure; these are retardation of the circulation of the blood, paleness and coldness of the face and extremities, smallness and tension of the pulse, diminution in the number of the heart's contractions, the latter symptom being present in most cases, and frequently to a remarkable degree. These phenomena may almost with certainty be explained as of a reflex nature, and dependent on irritation of the sensory nerves of the abdomen. *The influence in operation here, then, is reflex, the action of the heart being inhibited, by irritation of the medullary centre of the vagus*, as in Goltz's percussion experiment, and its later modifications. By means of these experiments (which have already been mentioned in the physiological part of this work) not only the disturbances of the circulation accompanying the visceral neuralgias have been in the most satisfactory way explained, but important evidence was adduced in support of the theory that these neuralgias have their origin in the sympathetic.

Besides the indications quoted above, Romberg brings forward another symptom as characteristic of the hyperæsthesias of the sympathetic, and to it we must, in a few words, refer. This is the subjective sensation of faintness and weakness, the sense of impending death, felt by the patient while the attack lasts, and which is observable in the sunken, anxious expression on the face, the compressed lips, the twitching of the nose, &c. We have already noticed this great subjective anxiety, which appears quite disproportionate to the severity of the objective phenomena in paroxysms of angina pectoris, and have explained it and the præcordial pain as being due to neuralgic irritation of the sensory fibres of the cardiac

nerves. Regarding colic and similar forms of visceral neuralgia, which have been described as hyperæsthesias of the solar plexus, coeliac neuralgia, &c., we would express ourselves in the same way, considering this symptom a neuralgic, radiative phenomenon, and not the reflex consequence of the embarrassed action of the heart. In this case as in the former, the latter theory is opposed by the fact that obstructions of the circulation of a much more severe nature than ever occur in attacks of colic are frequently met with without any corresponding feeling of oppression; and this sensation may be present without any objective symptoms of disturbance of the circulation (paleness of the skin and a small and slow pulse) accompanying the attack. We ourselves have observed this sense of oppression in great severity in persons whose face and extremities remained red during the seizure, whose heart continued acting powerfully, and in whose pulse there was no diminution in volume.

There still remains for discussion the question, usually quite ignored, regarding the route by which the painful impressions are conveyed to the sensory centre. We reject the theory of transference of impressions, "*Querleitung*," defended by Küttner (on pathological grounds) and by Volkmann; we feel inclined rather to accept it as a necessary postulate, confirmed by anatomical facts, that there is a continuous connection through the *ramicommunicantes*, between afferent sympathetic fibres and filaments in the posterior roots and posterior columns. Under this presupposition we think it not improbable that, at least for the most part, the painful enteralgic sensations reach the sensorium through the splanchnic nerves.

As we mentioned in the physiological section of this paper, Ludwig and Haffter, and Nasse, in their experiments on animals, found the splanchnic nerves to a high degree sensitive to painful impressions. Another symptom is perhaps of weight as showing that the splanchnic may be involved in this disease, namely, the extreme constipation that almost always attends colic, especially lead colic. This cannot be explained either by the inconstant and transitory spasmodic action or by paralysis (*Mérait*) of parts of the intestine. It is more probably to be traced to increased action of those filaments of the splanchnic which, when irritated, arrest the peristaltic movements of the small intestine.

Besides the sensory nerves of the intestines distributed in the splanchnic, perhaps *sensory fibres* from the plexuses

surrounding the abdominal arteries may be involved in the enteralgic attack. We are here reminded of Colin's experiments; he showed that the arteries of the abdominal viscera were possessed of great sensibility, in which the arteries of other parts were wanting.

Against our theory concerning the share borne by the splanchnic, it may be said that v. Bezold could not, by irritating the splanchnic nerves in mammalia, succeed in producing the same effects (arrest of the heart's movements by reflex action) that followed irritation of the mesenteric fibres in frogs. The negative result of these experiments allows only one conclusion to be drawn, that besides the splanchnic nerves other afferent trunks transmitting reflex influences may participate in the pathological irritation causing the attack of colic—perhaps the nerves which accompany the mesenteric artery.

II.—*Hyperæsthesia of the Solar Plexus.*

Neuralgia Celiaca.

Under the above name Autenrieth and Romberg included certain phenomena which, in their opinion, closely resemble the vagus-neuralgias of the stomach (gastrodynia neuralgica), but which proceed, not from the vagus, but from the solar plexus of the sympathetic. For the details of symptoms we must refer to Romberg's masterly description of this disease; we would only mention that the pain, as in gastric neuralgia, is felt chiefly in the epigastrium, radiating towards the back or the chest.

Romberg believes the cardialgia accompanying intermittent fever, described by Borsieri,* to be of this nature.

With regard to diagnosis, Romberg remarks, "The feeling of weakness accompanying the pain, the sensation of impending dissolution, which is distinctly observable in the circulation and general aspect of the patient, appears to me to be the symptom which is pathognomonic of celiac neuralgia, and which defines it clearly from neuralgia of the vagus." We have already fully discussed the sensation of oppression supposed to be characteristic of the sympathetic neuralgias, and its relation to the disturbances of the circulation which frequently accompany it; we can here refer only to what has already been stated. The uncertainty in

* Borsieri, "Instit. med. pract.," vol. i., p. 235.

Romberg's distinction has been especially noticed by Henoch,* who affirms that the two affections, clinically and therapeutically, are almost identical. v. Bamberger also holds that Romberg's description of *coeliac neuralgia* is simply an account of the symptoms of an acute *cardialgic* attack, and, moreover, he has not been able to find any anatomical indication of structural change in the *coeliac plexus*. Volz, who has met with nineteen cases of this form of disease, found nothing abnormal on post-mortem examination of one of these cases except cancer of the pancreas.†

Wittmaack‡ opposes Henoch, and is in favour of Romberg's distinction; in addition to the specific feeling of faintness he instances several other diagnostic symptoms which, however, seem to us to be of no great importance, namely, that *coeliac neuralgia* does not usually occur in young persons, that it is found more seldom in connection with sexual disorders (irregularities in menstruation) and lasts a shorter time than *gastrodynia neuralgica*. Even if we admit all these considerations, they do not prove that we have to do with an affection of the sympathetic, and especially of the solar plexus.

There are no available pathological anatomical facts, and no adequate physiological grounds, to enable us to make a proper estimate of the nature of this neurose. Anatomy and physiological experiment certainly show that twigs from the solar plexus take part in the innervation of the stomach, but in no way prove that sensory nerves, having a reflex action on the stomach, come from the same source.

III.—*Hyperæsthesia of the Hypogastric Plexus.*

This form of sympathetic neuralgia, first noticed by Romberg, is characterised, according to him, by painful sensations in the lower abdominal and sacral regions, radiating to the upper part of the thigh and the parts supplied by the spinal hæmorrhoidal nerves. It is most common in the female sex, with hysteria or irregularities of menstruation, occurring often at the commencement of puberty. Of this nature are many of those symptoms to which the public and some

* Henoch, "Klinik der Unterleibskrankheiten," Berlin, 1854, Bd. ii., p. 184-186.

† v. Bamberger, "Krankheiten des chylopoëtischen Systems," Erlangen, 1855. Virchow's "Spec. Path. und Ther.," Bd. vi., 1te Abtheilung, p. 168.

‡ Wittmaack, "Pathologie und Therapie der Sensibilitätsneurosen." Leipzig, 1861, p. 242.

physicians apply the vague terms *menstrual colic* and, in men, *hæmorrhoidal colic*.

Romberg supposes the sympathetic nature of this complaint to be indicated by "disorders of the circulation and secretion in the organs affected," which he looks upon as *consequences* of the hyperæsthesia of the hypogastric plexus. It is, nevertheless, questionable whether the local disorders of circulation and secretion (especially those of menstruation) do not frequently precede the neuralgic phenomena, and stand rather in a *causal* relation to them. Authors, however, who believe in a "hæmorrhoidal dyscrasia" consider the neuralgia only as a later symptom of that affection. As regards specially the participation of the hypogastric plexus in this neurose we know far too little about its functions generally, and of its sensory function in particular, to be able to give a decision on the matter on physiological grounds.

IV.—*Hyperæsthesia of the Spermatic Plexus.*

Neuralgia Spermatica.

In this category Romberg includes those affections of the male sex which Sir Astley Cooper has described as "*irritable testis*," others as "*neuralgia testis*" or "*neuralgia spermatica*." Valleix* holds that it is identical with the "*neuralgia ileoscrotalis*" described by Chaussier,† and places its seat in the region of the cerebrospinal nerve trunks of the lumbar plexus. Leubuscher‡ also seems inclined to this belief; while Hasse,§ like Romberg, locates the affection in the spermatic plexus, and affirms that it is due to dilatation of the veins, with or without varicocele, in the tissue of the testicle. Cahen is of opinion that the disease in question is a primary genitocrural neuralgia, to which is frequently added, as in the other neuralgias, an affection of the vasomotor nerves. This he infers from the swelling and dilatation of the vessels of the testicle which, according to him, are not causal but secondary.

* Valleix, "Observations on the Structure and diseases of the Testis." London, 1830, p. 49.

† Chaussier, "Table Synoptique de la Neuralgie suivant la Nomenclature Méthod. de l'Anat." 1803.—Valleix, "Traité des Neuralgies ou Affections doul. des Nerfs." Paris, 1841.—See also Neucourt, "Archiv Gen.," July and August, 1858.

‡ Leubuscher, "Krankheiten des Nervensystems," Lpz. 1860, p. 83.

§ Hasse: "Krankheiten der Nervensystems" (Virchows "spec. Path. and Th.," Band IV., Abthl. 1), 2te. Aufl; Erlangen, 1868, p. 82.

In connection with neuralgia testis we may mention some other neuroses of the genital organs, as rising probably from the same or a neighbouring part of the nervous system. Amongst these are *neuralgia of the urethra*, met with only in men; the *sensation of sexual pleasure exaggerated till it becomes a hyperæsthesia*, occurring most commonly among women, rarely among men, and the disease described by Gooch* as *irritable uterus*, and by others as uterine neuralgia. Cahen,† with regard to the latter, thinks that there is an immediate connection between the neuralgia and other functional disorders of the female genital organs considered by him vasomotor phenomena. According to him we have to do with a primary ileo-lumbar neuralgia, to which is added a vasomotor neurose of the uterus (congestion, hæmorrhage), frequently accompanied by abnormal secretion. The reverse relationship, that the ileo-lumbar neuralgia follows the uterine disease, is, in his estimation, not consistent with the order in which the symptoms present themselves.

These authors, in describing such obscure groups of symptoms, have adduced nothing that would warrant us in localising the seat of these affections in any sharply-defined part of the sympathetic system; and physiology and pathological anatomy certainly furnish us with no adequate grounds for so doing.

XII.—ANÆSTHESIAS OF THE SYMPATHETIC SYSTEM.

The anæsthesias of the sympathetic, like the hyperæsthesias, have been erected into a special group of sensory neuroses. In the meantime it is merely an empty framework, waiting to be filled up at some future time. We will in no way dispute the possibility of their existence, but we cannot conceal our impression that every condition necessary for such a nosological arrangement is absent. Physiological experiment and the results of pathological investigation have so far provided us with no such data, and we possess no trustworthy functional tests by means of which we can diagnose diminution of sensibility in the sympathetic; on the organs supplied by the sympathetic there is conferred, under normal circumstances, a very slight, almost imperceptible degree of sensibility, and this entirely in the indefinite

* Gooch, "Account of Some of the most Important Diseases Peculiar to Women." London, 1831, p. 299.

† Cahen, "Des Névroses Vasomotrices." Arch. Gen., 1863, T. ii.

form of the so-called *common sensation*, while muscular sensation and all the special qualities of the sense of touch are denied to the "vegetative" organs of the body. Thence we are asked to infer, improbable as it may appear, that the quantitative diminution of this almost imperceptible common sensation makes itself clearly known to our consciousness, both subjectively and objectively, according to the analogies presented to us by the other anæsthesias! In this difficulty the extensive reflex connections of the sympathetic have been appealed to, and it has been attempted to explain anæsthesia of the sympathetic as due to the arrest or diminution of regular reflex action (as in the peristaltic movements of the intestines). But a double objection is here met with. First, with reference to most of the motor phenomena, those of the intestines for example, it is still very problematical whether, under normal conditions, they are really reflex, and not rather produced principally or exclusively by direct automatic stimulation of peripheral ganglionic apparatuses; secondly, it does not necessarily follow from the absence of habitual reflex movements, even when the motor connection is shown to be complete and perfect, that we have to do with an actual anæsthesia, *i.e.*, with a condition in which sensory impressions are enfeebled during transmission through, or are absolutely arrested in, the sensory nerve-trunks. Absence of reflex phenomena may be due to disturbances in the central part of the track in which the reflex influence travels, in those nervous apparatuses (ganglionic cells) in which the afferent sensory stimulus is transformed into a motor impulse; and these central affections may occur notwithstanding the complete integrity of the nervous channels by which the sensory impressions are conveyed. Reflex action may thus be diminished or even arrested both while sensibility is intact and under the opposite conditions; this is sufficiently proved by many cases of *Tabes dorsalis*, and other well-known examples.

XIII.—*SYMPATHETIC PARALYSES AND SPASMODIC AFFECTIONS OF VOLUNTARY MUSCLES.—REFLEX PARALYSIS, DIPHTHERITIC PARALYSIS, &c.—TABES DORSALIS (ATAXIE LOCOMOTRICE PROGRESSIVE).—EPILEPSY.*

In the preceding parts we have dealt with many motor diseases of a paralytic or spasmodic character, which appear to be the result of the pathologically changed *innervation of unstriped muscular fibres* on the part of the sympathetic

system. We have seen mydriasis rise from a state of spasm in the sympathetic pupillary fibres, and myosis from paralysis of the same; we have shown how it is probable that the exophthalmos and other symptoms of Basedow's disease are caused by a change in the action of the sympathetic on the unstriated muscles of the orbit; we have discussed, when considering angina pectoris, the dependence of motor anomalies in the heart and blood vessels on the sympathetic nerve; and, finally, we have repeatedly pointed to the motor connections of the sympathetic with abdominal organs (the intestinal canal, bladder, genital apparatus, &c.) as the source of the pathological motor disturbances in these organs. We could throw only a few gleams of light on these conditions, and on the circumstances that warranted us in including them in the "pathology of the sympathetic system," because it has not been hitherto possible to fix definitely the course of the motor innervation of the organs in question, to separate sufficiently from each other the relations of this course to some parts of the sympathetic nervous system and to other (cerebro-spinal) nerves, and to make it possible to differentiate clinically between the pathological affections arising therefrom. We know, for instance, that the majority of the direct motor fibres of the bladder proceed from the spinal "*centrum genito-spinale superius*" and the "*centrum inferius*" through the sacral nerves, others, however, passing through the sympathetic (hypogastric plexus) to the vesical plexus. How is it, then, possible for us to distinguish diagnostically between a paralysis of the sympathetic and one of the spinal motor nerves of the bladder, and thus to speak of a sympathetic paralysis of the bladder, properly so-called? Such is the case also with regard to the intestines, uterus, &c., as has been formerly remarked when considering the motor innervation of these organs. When Romberg, as in the case of the hyperæsthesias and anæsthesias, made a special group of the paralyzes of the muscles supplied by the sympathetic trunks, and included in it paralysis of the nerves of the heart, pharynx, and œsophagus, he was justified in so doing by the earlier views regarding the motor innervation of the parts, and especially by the Bidder-Volkmann theory of the independence of the sympathetic system. Now, however, such an assumption would but imperfectly correspond to the state of physiological knowledge. Though our present information does not enable us to recognise as such the sympathetic paralyzes and states of spasm in the smooth

muscular fibres, and practically to distinguish between these and motor neuroses of other nerves, there can be no doubt as to their theoretical title to recognition, in spite of the oft-quoted results of experimental physiology. Even now, notwithstanding the above difficulties, we may with certainty state that destruction of certain parts of the abdominal sympathetic nerve must result in paralysis of the intestine, bladder, female genital apparatus, &c.; that, on the other hand, paresis and paralysis of the abdominal organs may depend on loss of the conducting power and irritability of branches, plexuses, ganglia, and trunks of the sympathetic nerve.

Apart from the neuroses connected with the smooth muscular fibres and the so-called vegetative organs, many experiments have been made with the view of claiming for the sympathetic an important rôle in the production of certain forms of motor disorder in the *striped voluntary muscles*.

We now specially refer to those paralyzes occurring in connection with certain affections of the abdominal organs (the intestines, the urinary and sexual apparatuses) which appear to have been known to Willis,* and which were named *Sympathetic Paralysis* by Whytt† and Prochaska,‡ and *Reflex Paralysis* by Romberg.§

The latter at first started the theory, founded on the experiments of Combaire || (who observed paralysis of the hinder extremities of animals after extirpation of the kidneys), that *by decrease or absence of the centipetal conducting power in sympathetic nerve trunks* a genuine paralysis may be brought on in parts supplied by spinal motor nerves. This theory he withdrew in a later edition of his book, as without physiological support.¶ Romberg especially included among the paralyzes produced in the above way the

* Willis, in explanation of this, draws attention to the anatomical connection between the sympathetic and the nerves of the extremities. "Nervi enim mesenterii non tantum cum intestinis, stomacho, jecore aliisque visceribus communicant, sed etiam cum lumborum aliarumque partium nervis, et consequenter cum artuum." (De anima brutorum quæ hominis vitalis et sensitiva est; Op. Omn. Geneva, 1680.)

† Whytt, "Observations on the nature, causes, and cure of the disorders which are commonly called nervous, hypochondriac, or hysteric, &c." Edinburgh, 1765.

‡ Prochaska, "Institutionum physiologiæ humanæ," vol. i. et. ii. Wien, 1806.

§ Romberg, L. c., p. 165.

|| Combaire, "Dissert. sur l'extirpation des reins." Paris, 1803.

¶ Romberg. "Lehrbuch der Nervenkrankheiten," 3 Aufl., 1855, p. 913.

saturnine and hysterical paralyses. Brown-Séquard,* Graves,† Stanley,‡ Jaccoud,§ Lewisson,|| Leyden,¶ Feinberg,** have given other explanations of the occurrence of reflex paralysis, the discussion of which would be too lengthy for this paper.

We pass over also the doubtful influence of the sympathetic in certain forms of paralysis and spasmodic affections of the eye (*strabismus convergens* from relaxation of the external recti muscles, *exophthalmos* from spasm of the obliqui, &c.), which have already been shortly mentioned in the physiological part of this essay. A detailed account of these also would be out of place, as the theories in question, so far as their bearing is of a pathological character, could claim attention only in virtue of their historical value. The same may be said in general also of the doctrines of Remak, who, as we saw, believed that all the voluntary muscles of the body were under a tonic influence proceeding from the sympathetic, and expressed the opinion that "*besides spinal paralysis and spasm, we may expect to meet with both sympathetic paralysis and sympathetic spasm in the voluntary muscles.*" In these words lie the beginning, and, to a certain extent, the programme, of the important part played by the sympathetic system for a long time in the pathology of the motor neuroses, especially in the galvano-therapeutics of these neuroses. We do not consider it necessary to quote here all the motor disorders which, almost without any physiological ground for so doing, have been gradually brought into connection with the sympathetic, both diagnostically

* Brown-Séquard, "Lectures on the diagnosis and treatment of the principal forms of paralysis of the lower extremities." Philadelphia, 1861, p. 24. His results were negated by those obtained by Gull; "Guy's Hosp. Rep.," vii., 1861.

† Graves, "Leçons de clinique médicale," traduites par Jaccoud, 2 Ed. Paris, 1863.

‡ Stanley, "On irritation of the spinal cord and its nerves in connection with disease of the kidneys"—"Med. Chir. Transactions," xviii., 1833.

§ Jaccoud, "Les paraplégies et l'ataxie du mouvement." Paris, 1864, p. 353. Jaccoud, following up his "exhaustion" theory, called the reflex paralysis *neuro-paralytic paralysis*.

|| Lewisson, "Über Hemmung der Thätigkeit der motorischen Nervencentra durch Reizung sensibler Nerven." Richert's und du Bois-Reymond's "Archiv," 1869, p. 255-266.

¶ Leyden, "Über Reflexlähmungen," in Volkmann's "Sammlung klinischer Vorträge," Leipzig, 1870, No. 2.

** Feinberg, "Ueber Reflexlähmungen," Berliner klin. Wochenschrift, 1871, No. 44-46.

and electro-therapeutically. We will mention only a few details as examples :—

In a communication on *spasm of the facial muscles*, which contains much that relates to this subject, Remak* mentions, amongst others, certain cases of facial paralysis in which the electric irritability of the muscles and nerve-trunks was completely lost. He then says—"When the constant current is passed through the cervical sympathetic of the affected side, even if only for a short time, we observe the striking phenomenon that the irritability of the muscles on the same side returns, the tension of the muscles of the opposite side relaxes, although the muscles on the paralysed side do not again come under the control of the will, and the irritability of the paralysed nerve-trunk does not increase in the slightest degree. It seems to me that here we have a physiological secret—namely, the greatest probability that the sympathetic exercises a direct or indirect influence over the voluntary muscles—a theory which has already received support from what I have seen in lead colic and progressive muscular atrophy, in which the paralysed and atrophied muscles gain in bulk, irritability, and power to perform their office as soon as the cervical or dorsal part of the sympathetic, on the same or the opposite side, comes under the influence of the current." There is no absolute proof, however, that in these cases the cervical or dorsal part of the sympathetic was acted on by the current, and the effects of the supposed galvanisation of the sympathetic do not correspond with the known physiological powers of that nerve, nor can any functional analogue be found for them.

Remak also mentioned a case of facial spasm which improved under treatment by galvanism, by placing the positive electrode at the level of the fifth cervical transverse process "on the spot where one might expect to find the ganglion medium of the sympathetic nerve." He supposes that there are two possible ways of explaining this fact; the first is "that the cause of the spasm is situated also in that part at which the cure was effected"—i.e., in the ganglion cervicale medium; the second that we have to do with an "indirect, catalytic influence," exercised, in his opinion, through a communicating branch between the ganglion medium and the ganglion thoracicum superius, which accompanies the vertebral artery in the vertebral

* "Berliner klin. Wochenschrift," 1864., No. 21-23.

canal and thus regulates the supply of blood to the base of the brain. Besides this form of spasm indirectly connected with the sympathetic system there are "genuine sympathetic facial convulsions—i.e., on the affected side of the face there is a peculiar variety of paralysis associated with spasmodic movements occurring in connection with unmistakable disease of the cervical parts of the sympathetic." Proof of the existence of this affection is still wanting.

In another place Remak * states concerning the dependence of diphtheric paralysis on change in the sympathetic, that he believes that it is caused by an affection of the ganglion cervicale superius, occurring with the diphtheritis. This theory of diphtheritic paralysis enabled him to "overcome all the paralytic phenomena by galvanisation of the ganglia superiora of the cervical sympathetic." He is of opinion that the tumefactions found by Bretonneau, Maingault, Trousseau, &c., in the neighbourhood of the angle of the lower jaw did not proceed from the lymphatic or salivary glands, but were caused by infiltration of the connective tissue, whereby the sympathetic also was affected—a hypothesis which is certainly not impossible, but one the truth of which still remains to be proved.

In another paper, written shortly after the last-mentioned, Remak† characterises as a "*dental neurose of the heart*" a condition in which the prominent symptoms were *trismus* and a *neurose of the heart* (accelerated pulse). In order to determine whether it was in the cervical sympathetic or in the vagus that the neurose originated, the constant galvanic current was tried; and as the passing of the current through a spot situated behind the angle of the lower jaw resulted in a fall in the frequency of the pulse, it was concluded that the ganglion cervicale superius was the part at fault. Remak, therefore, propounds the theory that the peripheral stimulus, when the cerebral activity is unaffected, produces spasm only when at the same time a ganglionic part of the sympathetic partakes in the irritation. In cases also of the convulsions of teething in children, so far as they are not of a meningitic origin, he considers it necessary to pay special heed to the occurrence of "swellings in the course of the cervical sympathetic." In these examples, which belong to the latest years of Remak's labours, are many brilliant and suggestive conceptions; they want, however, a physiological and pathological foundation. We thought it necessary to mention

* Remak, "Berl. klin. Wochenschrift," 1865, No. 13.

† Remak, "Berl. klin. Wochenschrift," 1865, No. 25.

them, as they exercised for a long period a most important influence; even at the present day, especially as regards galvano-therapeutics, they meet with extensive approbation.

We would here shortly discuss the relation stated by many authors to exist between the *sympathetic system* and the group of symptoms known as *Tabes dorsalis*, or *progressive locomotor ataxy*. Duchenne* has stated that in a number of cases of ataxy the cervical sympathetic must be regarded as the starting point of the morbid processes. This he infers from the frequent occurrence of the well-known oculo-pupillary symptoms—contraction of the pupil with increased vascularity and temperature of the eye, sometimes also dilatation of the pupil during the attack of pain, contraction and dilatation by turns without vascularity, or unilateral or bilateral myosis.

Duchenne explains in a somewhat far-fetched way the absence of positive post-mortem evidences in the sympathetic by the existence of degenerative changes in the posterior columns. He supposes that the sympathetic is in the first place *functionally* affected—thence the pupillary phenomena, increase of heat, &c.; that this “functional disturbance” in the sympathetic then exerts a “neuromparalytic action” on the posterior columns, thus giving rise to the atrophy.† The sudden attacks of pain in the bladder and rectum, and the disorders of the genital functions, which occur in some cases, are regarded by Duchenne as functional disorders originating in the abdominal sympathetic. He leaves quite unanswered the important question why we should not rather consider the pupillary, vasomotor, and urogenital centra situated in the spinal marrow as the points of origin of the functional anomalies connected with atrophy. Carré‡ and Friedreich examined some cases of progressive locomotor ataxy most carefully, and could discover no trace of change in the sympathetic. In one case of grey degeneration of the posterior columns and atrophy of the posterior roots, Westphal§ noticed nothing abnormal in the spinal

* Duchenne, “Recherches cliniques sur l'état pathologique du grand Sympathique dans l'Ataxie Locomotrice Progressive.” *Gaz. Hebdomadaire*, 1864, Nos. 8 and 10.

† Duchenne, “Sitzung der Acad. des Sciences, vom 18 Jan., 1864.

‡ Carré, “Nouvelles Recherches sur l'Ataxie Locomotrice Progressive.” Paris, 1865. “*Gaz. des Hôp.*,” 1865, No. 73.

§ Westphal, “*Virchow's Archiv*,” Band 39, p. 114.

ganglia; and in another* case he found no change in the cervical part of the sympathetic, or the uppermost cervical ganglion, both when examined fresh and after being hardened by treatment with turpentine. Vulpian's† researches had the same decidedly negative result; in very advanced cases of tabes he could demonstrate no pathological alteration, either in the trunk of the sympathetic or in any of its ganglia. However, if one believes in Duchenne's doctrine of "functional disturbance in the sympathetic," pathological anatomical proof of lesion of the sympathetic in tabes would clearly be superfluous. Remak,‡ in these cases, designated by him tabes dorsalis in its strictest sense (as opposed to tabes basalis, cervicalis, &c.), observed also inequalities in the pupils, taking the form of unilateral or bilateral dilatation with imperfect reaction when stimulated by light. He interprets these phenomena by the results of Brown-Séquard's and his own experiments, in which division of the sympathetic trunk below the upper lumbar ganglion, or of the branches of the celiac plexus (in frogs and mammalia), at once brought about a persistent dilatation of the pupil on the side operated on.

Remak also describes a special form of tabes, *tabes sympathetica* (identical with the so-called hysterical tabes), which occurs almost exclusively in the female sex. From the report§ of one such case, which was also associated with sympathetic paralysis of the face, we quote only the diagnosis:—"Neuroanglitis sympathica progressiva sexualis, with consecutive Ischæmia of the spinal cord;" we were unable to find in it any further specific information or anything characteristic of this form of disease.

The relation of *epilepsy* to the sympathetic system is still very obscure. On the whole the view formerly advanced by us appears lately to have gained ground—that many cases, especially of so-called peripheral epilepsy, are of an angioneurotic nature, and owe their origin partly to a direct and partly to a reflex irritation of vasomotor nerves.

Benedikt|| states "that the epileptic attack is primarily

* Westphal, "Virchow's Archiv," Band 39, p. 365.

† Vulpian, "Archives de Physiologie Normale et Pathologique," 1869, ii., p. 221.

‡ Remak, "Neue Beiträge zur Lehre von der Tabes," "Berl. klin. Wochenschrift," 1864, No. 41.

§ Remak, *ibidem*, p. 397.

|| Benedikt, "Zur Lehre von der Localisation der Epilepsie." Allgemeine Wiener med. Zeitung, 1870, Nos. 35 and 36.

caused by sudden spasm or relaxation of the vessels, and presents the most complete analogy to neuralgic attacks, only that here the irritation affects chiefly vasomotor nerves, and so leads directly or indirectly to anæmia or hyperæmia of the brain." He also thinks himself justified in assuming that the hippocampus major, indicated by Meynert as the part affected in epilepsy, is a vasomotor centre, irritation of which, whether from the cerebral hemispheres or by reflex influences from the periphery, induces the phenomena of the epileptic seizure.

Nothnagel holds, on the strength of his formerly-cited experiments, that the cervical sympathetic has a certain control over the arteries of the pia mater, contraction of which is accompanied by contraction of the arteries of the brain, which have the same origin; and he therefore believes that the epileptic seizure is the result of the anæmia of the brain consequent on the reflex contraction of its vessels. According to this theory the sympathetic plays a most important part in bringing on the epileptic attack, as most of the vasomotor nerves of the pia mater are included either in the cervical part of the sympathetic or in the ganglion supremum. Other investigators (Schultz, Riegel, Jolly) have not, however, confirmed the experimental grounds on which this doctrine rests.

XIV.—PATHOLOGICAL CHANGES IN THE SYMPATHETIC IN SOME INFECTIOUS AND OTHER DISEASES.

In this short additional note we group together some observations on changes in the sympathetic, to which no reference could be made in the former part of this work. These changes, which have hitherto been but slightly noticed, have been found chiefly in syphilis and other infectious diseases, in anomalies of the general nutrition (dyscrasias), &c.

Petrow* was the first who described the changes in the sympathetic in constitutional syphilis. In twelve cases in which he examined the cervical, thoracic, and solar plexuses, he found distinct alteration of the nerve elements and the interstitial connective tissue. In the first were pigmentary and colloid degeneration; associated with it was an affection of the endothelium surrounding the nerve-cells, characterised by great increase in the size and proliferation

* Petrow, "Die Veränderungen des Sympathischen Nervensystems bei Constitutioneller Syphilis." Virchow's "Archiv," 1873, Bd. 57, p. 121.

of the endothelium cells, and by secondary fatty metamorphosis of the same. The change in the interstitial connective tissue of the sympathetic consisted of hyperplasia, leading to sclerosis and atrophy of the nerve elements.

That these lesions of the sympathetic nervous system are really the result of syphilis is shown—apart from the fact of its occurrence in *all* the twelve cases examined—by their analogy to those pathological forms by which syphilis declares itself in other organs. Among these are especially the above-mentioned hyperplastic processes in the interstitial connective tissue.

Soon afterwards Pio Foà* wrote concerning some changes he had observed in the sympathetic in various diseases. These were found chiefly in the cervical and abdominal ganglia, and consisted sometimes of simple or fibrous atrophy, at other times of hyperæmia, sclerosis, pigmentary and fatty infiltration, amyloid degeneration, accumulation of colourless blood corpuscles, and the presence of micrococci in the blood-vessels of the ganglia. These alterations are well-marked in syphilis, Leukæmia, a high degree of cachexia, Pellagra, Tuberculosis, cardiac disorders, and infectious diseases. With respect to the affections of the heart, we may shortly state that de Giovanni† has lately directed attention to the participation of the sympathetic in organic cardiac diseases. He found in three cases contraction of the pupil, which he regards as the result of a venous stasis in the neighbourhood of the cervical sympathetic, which compresses the pupillary fibres; at the autopsy of one of these patients pathological changes were noticed in the cervical sympathetic.

In conclusion we would mention two reports by Köster‡ on the state of the cervical sympathetic in persons who had died suddenly from sunstroke. In one case, that of a soldier, the ganglion supremum of the right sympathetic was twice the size of the left, and was the seat of hæmorrhagic effusion; microscopically, the nerve-fibres were seen to be separated and disintegrated. There were smaller hæmorrhages in the lower, greater hæmorrhages in the upper, parts of the right sympathetic, while slight effusions of blood were found in and round both vagi and in the sheaths of both phrenic nerves. In the second case, that of a woman 21 years of age, the pathological phenomena were a

* Pio Foà, "Sull' Anatomia del gran Simpatico." Bologna, 1874.

† De Giovanni, "Annali univ. Tebbr," 1875, p. 216.

‡ Köster, "Berliner klinische Wochenschrift," 1875, No. 34,

hæmorrhagic infiltration and enlargement of both ganglia suprema of the cervical sympathetic, while the microscope revealed the same appearances as in the other case; there were also ecchymoses as large as peas in both vagi. In the first case the patient had lived twenty-four hours, having a pulse so rapid that its beats could scarcely be counted; there was, however, no acceleration of the respiration. The sudden death might be accounted for by the effect of some paralysing influence on the vasomotor nervous system.

ERRATA.

- Page 1—Line 2nd from bottom: for "des," read "der."
 " 5—Line 5th from bottom: for "Nerven Krankheiten," read "Nervenkrankheiten."
 " 5—Line 2nd from bottom: for "Experimentelles," read "Experimentales."
 " 7—Line 7th from top: for "à," read "a."
 " 7—Line 4th from bottom: for "Psysiologie," read "Physiologie."
 " 21—Line 2nd from bottom: for "f," read "T."
 " 22—Line 6th from bottom: for "Innervationsmenge," read "Innervationswege."
 " 24—Line 8th from top, and in subsequent parts of the paper: for "V. Bezold," read "v. Bezold."
 " 26—Line 3rd from bottom: for "1878," read "1870."
 " 33—Line 7th from bottom: for "Medecin und Naturwissenschaft," read "Medecin und Naturwissenschaft."
 " 34—Line 23rd from top: for "F. Rosenthal," read "J. Rosenthal."
 " 38—Line 10th from bottom: for "V. Graefe," read "v. Graefe."
 " 41—Line 22nd from top: for "evigentes," read "erigentes."
 " 41—Bottom line: for "Untersuchungen, Frankfort," read "Untersuchungen, Frankfurt."
 " 42—Bottom line: for "vendus," read "rendus."
 " 44—Line 9th from bottom: after "Heft," insert "2."
 " 47—Line 9th from top: for "Coates," read "Coats."
 " 50—Line 20th from top: for "choroidea," read "choroideæ."
 " 50—Line 3rd from bottom: for "Wienam," read "Wien am."
 " 54—Line 22nd from top, and in subsequent parts of the paper: for "Bäerwinkel," read "Baerwinkel."
 " 58—Line 12th from bottom: for "Séguin," read "Seguin."
 " 59—Line 19th from bottom: for "Hemicrania," read "Hemicrania."
 " 60—Top line: for "Hyperæsthesia," read "Hyperæsthesia."
 " 63—Line 4th from bottom: for "on," read "ou."
 " 69—Line 3rd from bottom: for "polarne," read "polaren."
 " 70—Top line: for "Trommhold," read "Frommhold."
 " 80—Line 3rd from top, and page 92, line 7th from bottom: for "Friedrich," read "Friedreich."
 " 84—Line 7th from bottom: for "Spielman," read "Spielmann."
 " 84—Line 4th from bottom: for "Strasbourg," read "Strassbourg."
 " 84—Line 2nd from bottom: for "Aertyte in Wein," read "Aerzte in Wien."
 " 86—Line 4th from bottom: for "a," read "ac."
 " 87—Line 4th from bottom: for "comtes," read "comptes."
 " 88—Line 2nd from bottom: for "Ophthalmogie," read "Ophthalmologie."

- Page 98—Line 15 from bottom: *for* "pathognomic," *read* "pathognomonic."
 " 98—Line 12 from bottom: *for* "sterum," *read* "sternum."
 " 105—Line 13 from bottom: *for* "sardaic," *read* "cardiac."
 " 106—Line 7 from top: *for* "ramicommunicantes," *read* "rami communi-
 cantes."
 " 117—Line 6 from top: same error.
 " 125—Line 20 from bottom: same error.
 " 110—Line 12 from bottom: *for* "splachninc," *read* "splanchnic."
 " 114—Line 11 from bottom: *for* "neurilema," *read* "neurilemma."
 " 114—Lines 17 and 22 from top: same error.
 " 117—Line 12 from top: *delete* "is."
 " 121—Line 24 from bottom: *for* "intestinal," *read* "abdominal."
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